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Variability of Graphomotor Task Performance in Adults with ADHD: A Kinematic Approach

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by

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Variability of Graphomotor Task Performance in Adults with ADHD: A Kinematic Approach

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ABSTRACT

Attention-Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by symptoms of inattention and/or a combination of hyperactivity and impulsivity. Motor problems, including poor graphomotor skills, are frequently found in those with ADHD and have been noted to be undertreated. Variability of performance within several domains has also been indicated as a hallmark of ADHD. The present study sought to 1) determine whether the variability of performance observed in other psychological domains in those diagnosed with ADHD manifests within kinematic variables of graphomotor output and 2) determine whether a novel writing task differentially affects the graphomotor output of adults diagnosed with ADHD versus controls. Findings and implications are discussed.

Keywords: digitizing tablet, stimulant medication, fine motor skills, variability of task performance

DEDICATION

To my wife Laura, without whose love and support, my current path would be unbearable.

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CHAPTER I

INTRODUCTION

Attention-Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized primarily by symptoms of inattention and/or a combination of hyperactivity and impulsivity (Barkley, 2006). In addition to the diagnostic criteria that define ADHD, several other impairments have been consistently identified in those with ADHD. These characteristics include motor skill impairments, such as poor handwriting, and variability of task performance, which manifests within several domains. One promising method that has been used to investigate graphomotor functioning (i.e., handwriting) is kinematic analysis, which has historically involved the use of digitizing technology. Kinematic analysis of graphomotor functioning in the ADHD population has indicated that within the context of medication status (i.e., whether taking prescribed dosages of stimulant medication or having discontinued medication), children with ADHD differ in automatized graphomotor fluency when compared to unaffected children. Similar results have not been documented in adults with ADHD. However, no study has investigated whether the variability of performance that is observed within the ADHD population extends into the graphomotor domain.

CHAPTER II

REVIEW OF LITERATURE

Diagnostic Criteria

The most recent version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) estimates the prevalence of ADHD to be between 3% and 7% of school aged children in the United States. In adults, the prevalence of ADHD has been estimated at approximately 4% (as cited in Biederman, 2005). Data demonstrating persistence of ADHD symptomatology from childhood into adulthood are mixed, with estimates ranging between 4% (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998) and 85% (Barkley, Fischer, Smallish, & Fletcher, 2002). Although estimations may be conservative in general (Root & Resnick, 2003), Barkley (2006) has indicated that prevalence estimates of ADHD differs based on a variety of factors, including sex, age, diagnostic criteria, data collection methods, and country of origin.

Utilizing criteria described in the DSM-IV-TR, a diagnosis of ADHD can be given to those who demonstrate either "six (or more)" symptoms of inattention and/or "six (or more)" symptoms related to hyperactivity and impulsivity that "have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level" (American Psychiatric Association, 2000, p. 92). Further, these symptoms must have been observed before the individual was 7 years old and with impairment occurring in two or more settings (e.g., at school, in the home, and/or in the work-place). Specific subtypes of ADHD, which correspond to different combinations of symptomatology, include ADHD combined type (ADHD-C), ADHD Predominantly Inattentive Type

(ADHD-PI), and ADHD Predominantly Hyperactive-Impulsive Type (ADHD-HI). A diagnosis of ADHD-C requires that both six or more symptoms of inattention and six or more symptoms of hyperactivity-impulsivity have been present for at least the past six months. The ADHD-PI subtype is indicated when six or more symptoms of inattention are present for at least six months, but fewer than six symptoms of hyperactivity-impulsivity are present during this same time period. Finally, a diagnosis of ADHD-HI is appropriate if six or more symptoms related to hyperactivity-impulsivity have been present for at least the past six months, but fewer than six symptoms of inattention are present during this same time period.

Etiology of ADHD

The etiology of ADHD is complex in nature, although recent research implicates neurological and genetic factors as primary agents of pathogenesis (Barkley, 2006). The advent and subsequent popularity of modern neuroimaging techniques such as magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI) have enhanced the ability of researchers to analyze the structural neuroanatomy of individuals in a non-invasive manner. In the case of ADHD, several consistent findings have emerged with regard to abnormal structure of the central nervous system (Barkley, 2006). Widespread reductions of cortical gray matter have been found in the frontal, parietal, temporal, and occipital lobes of the cerebral cortex in general (Batty et al., 2010; Shaw et al., 2006) and in frontal and posterior association cortices in particular (Narr et al., 2009). Although findings vary to some degree between studies, reductions in gray matter volume have been found in more circumscribed areas of the cortex and subcortical nuclei in both children and adults with ADHD. These areas include the prefrontal and dorsolateral

prefrontal cortices, basal ganglia (caudate nucleus, globus pallidus, putamen, and substantia nigra), and anterior cingulate cortex (Amico, Stauber, Koutsouleris, & Frodl, 2010; Castellanos, Geidd, Marsh, & Hamburger, 1996; McAlonan et al., 2007; Romanos et al., 2010; Seidman et al., 2011). Reductions in the infratentorial structural volume of the cerebellar vermis have also been found in both children (Castellanos et al., 2001; Durston et al., 2004; Mackie et al., 2007) and adults (Seidman et al., 2011) diagnosed with ADHD.

Research also indicates that although those diagnosed with ADHD do not consistently demonstrate global reductions in white matter volume compared to controls (Amico et al., 2010; Batty et al., 2010; Durston et al., 2004; McAlonan et al., 2007; Narr et al., 2009), reduced white matter volumes in specific areas of the cerebrum have been more consistently documented. For example, McAlonan et al. (2007) found that white matter tracts of the corpus callosum evidenced reduced volume in those diagnosed with ADHD. This finding is consistent with past studies indicating reduction in white matter of the corpus callosum in general (Hynd et al., 1991) and the splenium of the corpus callosum in particular (Semrud-Clikeman et al., 1994). Other studies investigating the structural integrity of white matter pathways connecting different regions of the cerebrum suggest that these pathways appear to be compromised in the ADHD population (Konrad & Eickhoff, 2010). More specifically, the superior longitudinal fasciculus and anterior corona radiata, which are tracts projecting between the frontal cortex and basal ganglia, have evinced reduced white matter integrity in children and adults based on measurements of fractional anisotropy (FA; representing the directionality and shape of the water molecules within the tract), mean diffusivity (MD), and apparent diffusion

coefficient (ADC; representing the volume of white matter diffusion) (Liston, Cohen, Teslovich, Levenson, & Casey, 2011). Due to the aforementioned inconsistencies in white matter volumetric findings in the ADHD literature, however, firm conclusions concerning the role of white matter pathways in the pathophysiology of ADHD cannot be drawn at this time.

Although relationships between ADHD symptomatology and structural abnormalities can only be inferred due to the nature of these studies, functional neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), provide additional evidence that structures believed to subserve abilities related to attention, inhibition, and motor control – abilities that are impaired in those with ADHD – are the same structures that demonstrate structural abnormalities in ADHD (Brossard-Racine, Majnemer, & Shevell, 2011; Seidman et al., 2006; Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998; Shaw et al., 2006). Compared with healthy children, children with ADHD show abnormal patterns of activation (i.e., hypo-activation) in the prefrontal cortex, basal ganglia, and cerebellum when performing tasks related to attention, inhibition, motor control, and executive function (Bush et al., 1999; Durston et al., 2003; Posner et al., 2011; Rubia et al., 1999; Teicher et al., 2000; Vaidya et al., 1998; Yeo et al., 2003). Differences also appear to persist into adulthood. For example, Cubillo, Halari, Giampietro, Taylor, & Rubia (2011) found that compared with neurotypical individuals, medication naive adults diagnosed with ADHD in childhood who continued to demonstrate symptomatology into adulthood were found to have reduced activation in the orbital frontal cortex, medial frontal cortex, and striatum (i.e., basal ganglia) during tasks requiring inhibition, as well as reduced activation in the lateral

inferior and dorsolateral prefrontal cortices during tasks of working memory and attention.

Although studies have not demonstrated that ADHD occurs as a result of chromosomal abnormalities, several lines of research (i.e., family, adoption, twin, and genetic studies) indicate that ADHD has a high degree of heritability (Barkley, 2006). Highlighting the heritable and familial nature of ADHD – with some heritability rates estimated to be as high 0.76 (Faraone et al., 2005) – are findings that asymptomatic siblings of those diagnosed with ADHD demonstrate a trend towards similar volumetric reductions in cortical regions comparable with those found in their affected siblings (Durston et al., 2004).

At least seven genes appear to be implicated in the etiology of ADHD (Faraone et al., 2005), although several other genes are currently under investigation (see Banaschewski, Becker, Scherag, Franke, & Coghill, 2010, for a review). One example is the dopamine transporter gene, DAT1, which has received significant attention as mutations of this gene have been found to be related to presence of ADHD symptomatology in both adults (Brown et al., 2011) and children (Daly, Hawi, Fitzgerald, & Gill, 1999).

Dysfunction or imbalance of dopamine (DA), norepinephrine (NE), and noradrenaline (NA) neurotransmitters have also been implicated in the pathophysiology of ADHD (Arnsten, Berridge, & McCracken, 2009; Barkley, 2006; Biderman, 2005). In a recent review of the literature investigating the influences of DA and NA in ADHD, del Campo, Chamberlain, Sahakian, and Robbins (2011) suggested that DA and NA may play more specific roles in the presentation of ADHD symptomatology. That is, whereas

a combination of DA and NA abnormalities may affect functioning of the prefrontal cortex and by extension abilities related to inhibition, DA alone may affect functioning of subcortical structures such as the basal ganglia, in turn affecting attentional abilities.

Impairments Associated with ADHD

Beyond the primary symptoms of inattention, hyperactivity, and impulsivity, decades of research has demonstrated that ADHD is associated with numerous impairments affecting various domains of activities and functioning. Areas of impairment include cognitive functioning, language development and expression, motor skills, emotional regulation, academic performance, consistency of task performance, and general health and well-being (Barkley, 2006). Of particular interest here are the motor control problems, which are often under-treated in this population (Fliers et al., 2009), and the variability of task performance and expression.

Individuals diagnosed with ADHD have been shown to demonstrate variability in task performance and behaviours within several domains, including emotional expression (i.e., emotional lability; Barkley & Fischer, 2010; Posner et al., 2011), qualitative and quantitative handwriting production (Rosenblum, Epsztein, & Josman, 2008), in-phase bimanual coordination (Klimkeit, Sheppard, Lee, & Bradshaw, 2004), motor force output (Pereira, Eliasson, & Forssberg, 2000), and fine motor skill movements (Pitcher, Piek, & Barrett, 2002). Anecdotal reports from teachers and parents also suggest that children diagnosed with ADHD, as compared to healthy children, display a great deal of variability in their academic work and the quality with which they complete household duties (Barkley, 2006). Due to the observation that variability of task performance has been documented in several domains and appears to be ubiquitous in this population,

"some believe [variability of task performance] to be a primary deficit in ADHD" (Barkley, 2006, p. 136).

Although it is still unclear whether or not developmental motor milestones are generally delayed in children with ADHD (Barkley, 2006), the pervasive nature of motor difficulties that are observed in this population is highlighted by findings demonstrating significant comorbidity with Developmental Coordination Disorder, which is characterized by "marked impairment in the development of motor coordination" that "significantly interferes with academic achievement or activities of daily living" (American Psychiatric Association, 2000, pp. 56-57), when compared to the general population (Kadesjo & Gillberg, 2001; Piek, Pitcher, & Hay, 1999). Indeed, there is some evidence to suggest that ADHD and Developmental Coordination Disorder (DCD) share a genetic component (Martin, Piek, & Hay, 2006). Regardless of the presence of DCD, it is clear that those diagnosed with ADHD demonstrate motor impairments more frequently than the general population (Brossard-Racine, Majnemer, & Shevell., 2011). Examples of motor impairments found in those diagnosed with ADHD include poor handwriting (Brossard-Racine, Majnemer, Shevell, Snider, & Belanger, 2011); decreased speed and accuracy of complex (but not simple) fine and tactual motor performance (Meyer & Sagvolden, 2006); and deficits in balance, manual dexterity, coordination, and fine and gross motor skills (Piek, Pitcher, & Hay, 1999). Highlighting the importance of impairments in both motor functioning in general and timing of motor behaviour in particular are studies indicating that these problems are not only found in those diagnosed with ADHD, but also in siblings without an ADHD diagnosis. For example, Rommelse and colleagues (2008) found this relationship between affected and non-ADHD siblings

and concluded that based on the evidence, "Variability in motor timing appears a useful endophenotypic candidate: It is clearly associated with ADHD, it is also present in non-ADHD siblings, and it correlates within families" (p. 131). "Moderate" and statistically significant positive correlations between severity of ADHD symptomatology and severity of motor sequelae have also been documented (Rommelse et al., 2009), which provide additional support for the notion that both motor control dysfunction and variability in task performance could be considered as primary deficits in those diagnosed with ADHD.

Relevant to the academic success of children is the skill of handwriting. In a review of the literature investigating the handwriting skills of children diagnosed with ADHD, Brossard-Racine, Majnemer, Shevell, and Snider (2008) concluded that the handwriting of individuals in this population can be characterized as impaired, often illegible, and less organized than the handwriting of control children, which in turn results in low academic achievement. Poor qualitative writing observed in this population does not appear to be related to pure visual-perceptual, visual-motor integration, or linguistic difficulties; instead, poor performance likely involves many different processes (Brossard-Racine et al., 2008), including dysfunction in basic parameter setting, such as regulation of force, speed, and size of graphomotor movements (van Galen, 1991); motor control; and timing aspects of handwriting (Adi-Japha et al., 2007; Marcotte & Stern, 1997; Schoemaker, Ketelaars, van Zonneveld, Minderaa, & Mulder, 2005).

Kinematic Analysis of Handwriting

The volitional control of handwriting can be thought of as a complex process involving the integration of "cognitive, psychomotor, and biophysical processes" (van

Galen, 1991, p. 165) that are organized hierarchically and in parallel (Plamondon, 1995a) to produce meaningful visual-spatial output. Using a motor program metaphor, graphomotor processes begin with the retrieval of a high-level representation of the desired motor output - which might involve acquiring trajectory based stroke segments that can be combined to form complex symbols as opposed to retrieving whole letters or words stored within a visual-spatial "brain dictionary" (Lacquaniti, 1989, p. 287). This in turn is followed by a conversion of this representation into motor control "commands," finally ending with the neuromuscular system responding in the desired manner (Plamondon, Yu, Stelmach, & Clement, 1991). In addition, the neuromuscular and higher-order systems make necessary adjustments based on relevant "visual and/or kinesthetic feedback" (Dooijes, 1983, p. 104). Central nervous system structures likely involved in these motor output processes include the primary motor cortex, premotor cortex, supplemental motor area, basal ganglia, cerebellum, and spinal cord (Plamondon, 1995a).

Studies investigating the cognitive, psychomotor, and biophysical processes involved in graphomotor control generally support this process and its related components (Meulenbroek & Thomassen, 1993; Meulenbroek & van Galen, 1988; Portier & van Galen, 1992; Teulings, Thomassen, & van Galen, 1983; van Galen, 1990; Woch, Plamondon, & O'Reilly, 2011; see Plamondon & Maarse, 1989, for a review and evaluation of computational motor models of handwriting), with the initial phases of voluntary motor control represented by measurements of reaction time and the latter phases represented by measurements of total movement time (Bellgrove et al., 1997) and other variables. The use of objective tools and methods to assess handwriting movements

(e.g., kinematic analysis), then, can be viewed as a method to make inferences about these cognitive, psychomotor, and biophysical processes underlying graphomotor function.

Kinematic analysis involves the quantification of "time changes of position, velocity, and acceleration" (Viviani & Terzuolo, 1982, p. 431). Although many technological options are available for kinematic analysis, the use of digitizing tablets to capture handwriting signals has predominated in graphonomic research of both healthy and clinical populations over the past 30 years (for a review of early graphomotor research, including the use of digitizing tablets, see Graham & Weintraub, 1996). In the domain of graphonomics, kinematic measures can be quantified using parameters of time, acceleration, velocity, and pen pressure, and variables derived from these basic measures can be used to (a) describe abilities related to degree of movement automatization and fluency (Eichhorn et al., 1996; Margolin & Wing, 1983; Mergl, Tigges, Schroter, Moller, & Hegerl, 1999; Portier & van Galen, 1992; Teulings, Contreras-Vidal, Stelmach, & Adler, 1997; Yan, Rountree, Massman, Doody, & Li, 2008); (b) quantify the relative decelerations and accelerations of handwriting movements (Eichhorn et al., 1996; Mergl et al., 1999; Plamondon & Clement, 1991; van Galen, Portier, Smits-Engelsman, & Schomaker, 1993); (c) indicate stability, coordination, and consistency of an individual's handwriting (Mergl et al., 1999; Schroter et al., 2003; Teulings & Schomaker, 1993; Slavin, Phillips, Bradshaw, Hall, & Presnell, 1999); (d) indicate the sharing of processing resources, the difficulty of writing trajectories, and the presence of dysmetria (van Galen, 1991; Teulings, Contreras-Vidal, Stelmach, & Adler, 1997; Phillips et al., 2009); (e) quantify fine motor hypotonia and general proficiency (Mergl et al., 1999; Wann &

Nimmo-Smith, 1991; Phillips et al., 1999); and (f) indicate the smoothness and efficiency of movements (Bellgrove et al., 1997; Phillips et al., 2009). In this sense, the metrics produced by kinematic analyses of handwriting can be viewed as objective rather than subjective measurements of graphomotor performance.

Clinical Research Utilizing Kinematic Analysis

The use of digitizing technology to quantify graphomotor processes as an investigative and potentially diagnostic tool has been conducted with a multitude of patient populations. Pathologies and disorders investigated include, but are not limited to, ADHD (e.g., Adi-Japha et al., 2007; Flapper, Houwen, & Schoemaker, 2006; Schoemaker et al., 2005; Tucha & Lange, 2001, 2004, 2005; Tucha, Paul, & Lange, 2003); Dementia, Alzheimer's Disease, and Mild Cognitive Impairment (e.g., Bellgrove et al., 1997; Yan et al., 2008); DCD (e.g., Bo, Bastien, Kagerer, Contreras-Vidal, & Clark, 2008; Chang & Yu, 2010; Rosenblum & Livneh-Zirinski, 2008; Smits-Engelsman Niemeijer, & van Galen, 2001); Dysgraphia (e.g., Kushki, Schwellnus, Ilyas, & Chau, 2011; Overvelde & Hulstijn, in press; Rosenblum, Dvorkin, & Weiss, 2006; Smits-Engelsman & van Galen, 1997); Huntington's Disease (e.g., Phillips et al., 1996; Phillips, Chiu, Bradshaw, & Iansek, 1995; Slavin et al., 1999; Yaguez, Canavan, Lange, & Homberg, 1999); Learning Disability (e.g., Galli et al., 2011; van Roon, Caeyenberghs, Swinnen, & Smits-Engelsman, 2010); Schizophrenia (e.g., Grootens et al., 2009; Jahn et al., 2006; Putzhammer et al., 2005; Tigges et al., 2000); and Parkinson's Disease (e.g., Gangadhar et al., 2009; Poluha, Teulings, & Brookshire, 1998; Ponsen et al., 2006; Rand, Stelmach, & Bloedel, 2000; van Gemmert, Teulings, & Stelmach, 1998). Germane to the present study are findings related to ADHD.

Using qualitative variables such as legibility; spacing, letter size, and alignment consistency; organization of material within space; and letter insertions, transpositions, substitutions, and omissions, studies of handwriting produced by children diagnosed with ADHD indicate that their writing quality is generally poor, immature, and error-prone when compared with non-ADHD controls (Adi-Japha et al., 2007; Flapper et al., 2006; Lerer, Artner, & Lerer, 1979; Marcotte & Stern, 1997; Whalen, Henker, & Finck, 1981; Tucha & Lange, 2001). In addition, poor qualitative performance does not appear to be the result of purely linguistic, visual, perceptual, or visual motor integration deficits (Adi-Japha et al., 2007; Marcotte & Stern, 1997) and typically improves after taking prescribed dosages of stimulant medication (Lerer et al., 1979; Tucha & Lange, 2001; Whalen et al., 1981). Interestingly, kinematic analyses assessing objective, process related aspects of handwriting indicate that the handwriting produced by children diagnosed with ADHD is more dysfluent and thus appears less automatized when taking stimulant medication compared to when they are not taking prescribed medication, and is more dysfluent when such children are on stimulant medication than observed in controls (Flapper et al., 2006; Tucha & Lange, 2001, 2004, 2005). This pattern of fluency and dysfluency related to medication status, however, has not been observed in adults diagnosed with ADHD under similar conditions (Tucha & Lange, 2004). In these contexts, writing fluency is operationalized as the number of changes in direction of velocity or acceleration as recorded by digitizing technology and analyzed by appropriate software. Velocity profiles of fluent, automatized handwriting appear as smooth asymmetrical bell-shaped curves with few changes in velocity/acceleration direction, whereas dysfluent, unautomatized handwriting evinces velocity profiles with multiple “jagged peaks” and

many changes in the direction of velocity/acceleration. See Figures 1 and 2 for examples of fluent versus dysfluent vertical velocity profiles, respectively.

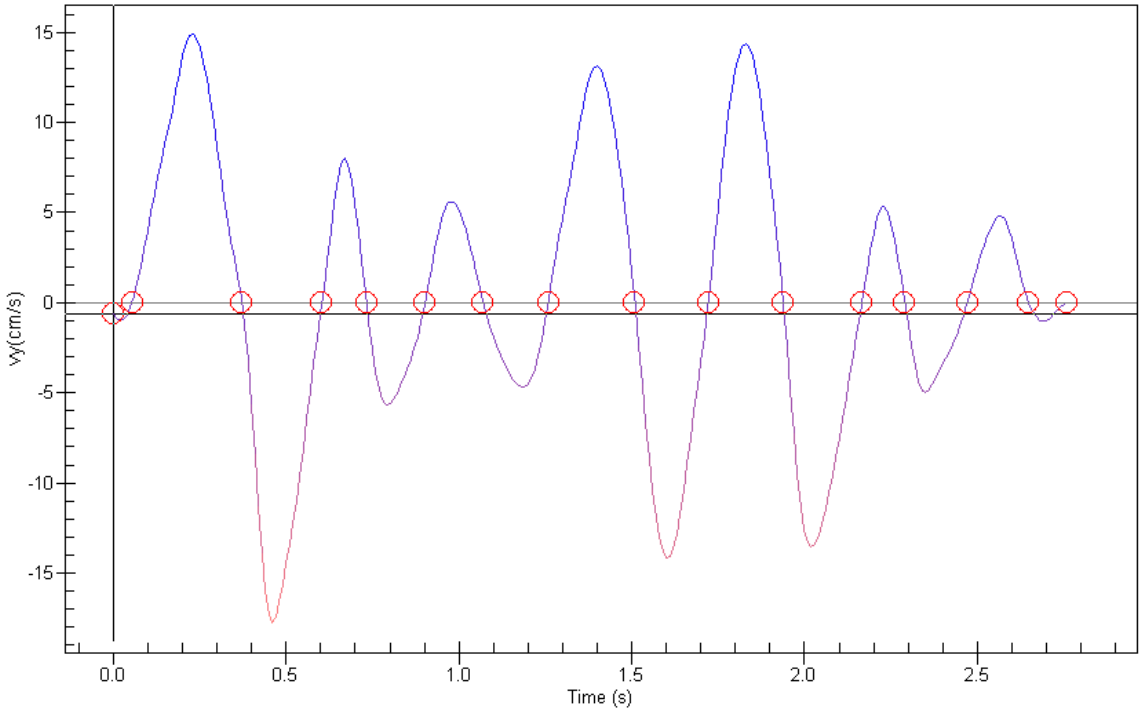


Figure 1. Velocity profile of the word "hello" written fluently.

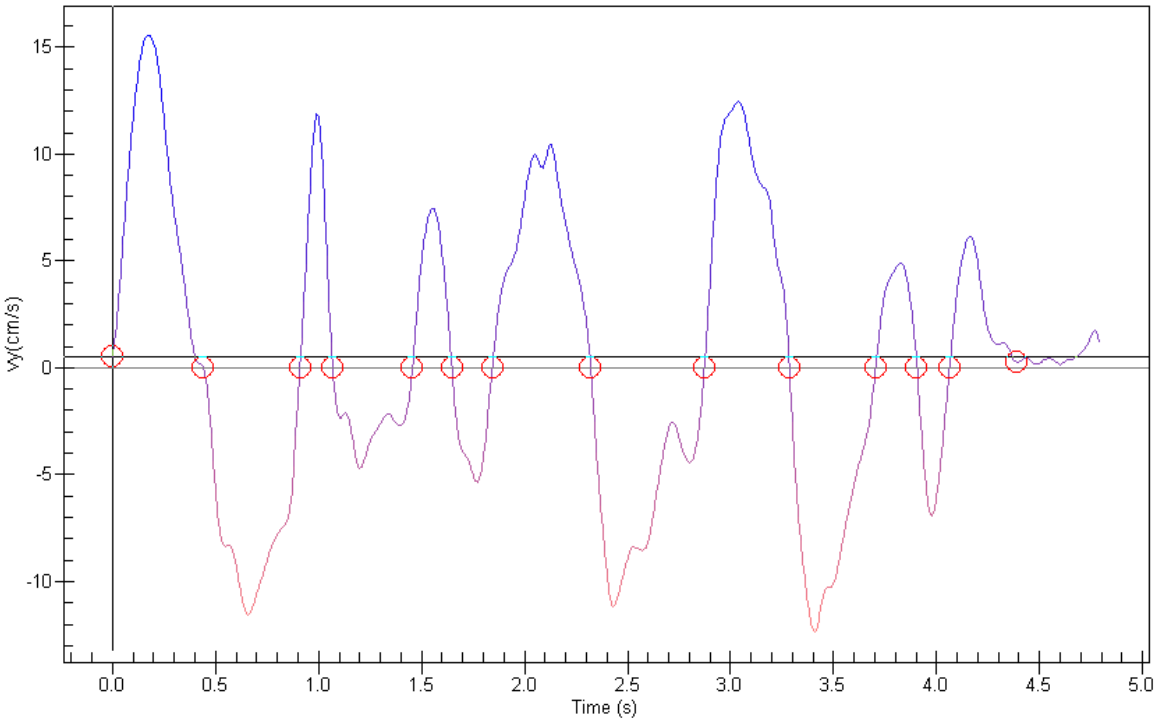


Figure 2. Velocity profile of the word "hello" written with simulated dysfluency.

In addition, these studies demonstrated that while off prescribed dosages of stimulant medication, kinematic measures of graphomotor fluency in affected children were not significantly different from those of non-ADHD controls. Further, it does not appear that these findings are due to a direct effect of medication, as fluent movements can be elicited from children with ADHD taking stimulant medication (Tucha & Lange, 2004). Rather, this decreased fluency and automaticity may be the result of a secondary effect resulting from enhanced attention, from greater cognitive control (Tucha & Lange, 2004; Tucha, Mecklinger, Walitza, & Lange, 2006; Tucha et al., 2003), or from possibly other cognitive, motor, or psychomotor processes influenced by stimulant medication. Alternatively, Lange et al. (2007) suggested that children and adults with ADHD may, in general, "have difficulties in skills whose acquisition starts as a [laboured] and conscious learning process that becomes automatic following consistent and frequent practice" (p.

256). Similarly, Flapper et al. (2006) noted that typically, accuracy is achieved before speed and fluency when learning a complex task. In turn, children with ADHD would first need to engage sufficient attentional resources and motor skills for an extended period of time before generating handwriting that is both fluent and accurate, noting that both attentional abilities and motor skills are reported to improve with methylphenidate treatment in adults, adolescents, and children diagnosed with ADHD (Bart, Podoly, & Bar-Haim, 2010; Lerer et al., 1979; Shafritz, Marchione, Gore, Shaywitz, & Shaywitz, 2004; Stray, Stray, Iverson, Ruud, & Ellersten, 2009; Tucha, Mecklinger, Laufkotter, et al., 2006; Tucha, Prell, et al., 2006).

The Present Study

There are few studies that have investigated the kinematic aspects of writing in adults diagnosed with ADHD, with no study specifically examining the potential variability of task performance within the kinematic aspects of graphomotor skills in adults diagnosed with ADHD, and no study comparing novel versus putatively automatized graphomotor processes in this population. As such, using a digitizing tablet to capture kinematic aspects of handwriting, the present study seeks to determine within the context of medication status 1) whether the variability of performance observed in other psychological domains (e.g., task persistence, emotion, and attention) in those diagnosed with ADHD manifests within kinematic variables associated with consistency, stability, and coordination during the execution of an automatized graphomotor task; and 2) assess the effects of novelty on consistency measures of graphomotor performance between adults with and without ADHD. Under the premise that handwriting output is generated from a velocity control perspective (i.e., that the central nervous system

produces volitional graphomotor output by controlling the velocity of an end-effector via interactions between higher-order cortical and sub-cortical systems and lower-level agonist and antagonist neuromuscular systems [Guerfali & Plamondon, 1997; Plamondon, 1993, 1995a, 1995b, 1998; Plamondon, Feng, & Woch, 2003]) and noting variability of performance/behaviour demonstrated in other psychological domains in those diagnosed with ADHD, it is hypothesized that 1) greater intra-individual variability in kinematic velocity measures will be observed in adults diagnosed with ADHD off medication when compared to neurotypical adults. Additionally, 2) although no a priori hypothesis is salient with regard to the effects of novelty on variability measures in those diagnosed with ADHD, it could be speculated that if variability of performance observed in adults with ADHD extends to the graphomotor domain, ADHD participants discontinuing medication will be differentially affected by a novel graphomotor task and in turn elicit greater levels of inconsistency compared to those without ADHD. Should statistically and practically significant differences become evident (i.e., differences of medium to large effect sizes), this would be the first study utilizing kinematic analysis to explicitly demonstrate variability of performance within the graphomotor domain in adults diagnosed with ADHD. Significant results indicating variability in kinematic performance would also add to the current literature indicating that ADHD is not simply a disorder of childhood, but rather, a disorder in which specific motor control differences extend into adulthood. Further, the results of this study would support conducting future research into the use of digitizing technology as an objective diagnostic and descriptive tool within the ADHD population, which in turn may enhance the specificity and/or sensitivity of current assessment and diagnostic techniques.

CHAPTER III

DESIGN AND METHODOLOGY

Participants

Power analysis (α = .05, [1 – β] = .80) indicated that using the proposed methodological design and statistical analysis, 52 total participants would be needed to detect a statistically significant difference of large effect size. For within-group differences, power analysis indicated that 16 participants would be needed to detect differences of large effect size.

Thirty-eight participants were recruited through three sources: control participants were recruited via the University of Windsor's Psychology Participant Pool (n = 31) and clinical participants were recruited through Student Disabilities Services (Education Development Center) at the University of Windsor and through the private practice of a local psychiatrist (n = 8). One control participant, however, requested that their data be removed from the study, resulting in a net of 30 control participants and 38 total participants. To minimize confounds related to extraneous visual and motor disturbances, participants included only those with normal or corrected to normal vision and those who did not have an existing neurological condition that would negatively affect graphomotor performance (e.g., cerebral palsy affecting the upper extremities, severe tendinitis, or carpal tunnel syndrome). In addition, clinical participants included only those who were currently taking prescribed dosages of stimulant medication for the treatment of ADHD symptoms. Participants recruited through the University of Windsor's Psychology Participant Pool received course bonus points (1 point for control participants based on one hour of participation time and 2 bonus points for clinical

participants based on two hours of participation time) for participating in the research study. Participants recruited through Student Disability Services at the University of Windsor and the private practice of the local psychiatrist received a $10 gift card and a chance to win one of two $50 debit cards via entry into a draw.

Materials and Apparatus

Demographic information and ADHD symptomatology. For the purposes of sample description, participant demographic information including age, sex, handedness, current medications (including type and dosage), ethnicity, official ADHD diagnosis and subtype (if applicable), and neurological status was collected from each participant via an in-person interview (see Appendix A for the interview form used). For participants diagnosed with ADHD, records pertaining to official diagnoses were reviewed and specific diagnoses if available (e.g., ADHD-C, ADHD-PI, ADHD-HI, and any comorbid diagnoses) were also recorded for descriptive purposes. In addition, all participants completed the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011). Based on DSM-IV diagnostic criteria, the BAARS-IV is a self-report questionnaire designed to evaluate current and/or childhood ADHD symptoms in adults between the ages of 18 and 81 years. According to the manual, the normative sample used to develop the BAARS-IV, which consisted of 1,249 adults between the ages of 18 and 96, "closely approximated the U.S. adult population based on the U.S. Census from the year 2000 concerning regional distribution, sex, race/ethnic group, marital status, employment status, total household income, and education" (Barkley, 2011, p. 14). After completion of the questionnaire, a total ADHD score, symptom count, and subscale scores for both current symptoms and childhood symptoms can be calculated. (Note: the BAARS-IV

also contains forms allowing current and childhood symptomatology scores to be derived based on reports from others through the use of an alternative quick-screen. These were not utilized in this study). The BAARS-IV also produces subscale scores related to four recognized ADHD symptom dimensions: Inattention, Hyperactivity, Impulsivity, and Sluggish Cognitive Tempo (SCT). According to the BAARS-IV manual, ADHD scores at or above the 93rd percentile may be interpreted as reflecting a significant abnormality and clinical significance in that domain. Because clinical participants participated in the research both on and off of their ADHD medication, they were asked to answer the questionnaire regarding their current symptomatology within the context of being off of their medication. Finally, if subtype identifier information was unavailable or unknown, a determination of subtype was made based upon the clinical participant's self-report current ADHD symptoms as measured by the BAARS-IV. That is, for clinical participants only, a subtype identifier of ADHD-PI was given if significant abnormality was reported only within the Inattention domain, a subtype identifier of ADHD-HI was given if significant abnormality was reported only within the Hyperactivity or Impulsivity domains, and a subtype identifier of ADHD-C was given if significant abnormality was reported within both the Inattention domain and the Hyperactivity or Impulsivity domains.

Internal consistency reliability of the BAARS-IV was reported by the manual to be "satisfactory" for current symptom total score and for each subscale/domain score for both current and childhood reported symptoms. Test-retest reliability was described as "reasonable" over a 2- to 3-week period. Finally, construct validity, discriminant validity, and criterion validity are reported to be "satisfactory." Internal consistency and

test-retest reliability measures for the BAARS-IV, as indicated by the manual (Barkley, 2011), are listed in Table 1.

Table 1

*Internal Consistency & Test-Retest Reliabilities of the BAARS-IV**

	Internal Consistency Reliability		Test-Retest Reliability**	
	Current Symptoms	Childhood Symptoms	Current Symptoms	Childhood Symptoms
ADHD Inattention	.902	.940	.66	.73
ADHD Hyperactivity	.776	.912†	.72	.82†
ADHD Impulsivity	.807		.76	
Total Score	.914	.947	.75	.79

Note. * Barkley Adult ADHD Rating Scale-IV. ** Test-retest reliability over a 2- to 3-week period. † Represents combined dimension of hyperactivity-impulsivity.

Estimate of intellectual ability. An estimate of IQ was derived using four subtests of the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV; Wechsler, 2008). Based on practical considerations and the best combination of short form reliability and validity coefficients (.953 and .940, respectively; see Sattler & Ryan, 2009), the four subtests used for estimating IQ were Block Design (BD), Vocabulary (VC), Arithmetic (AR), and Coding (CD). IQ estimates were used for descriptive purposes and for identifying initial group differences between the control group and the clinical group.

Kinematic analysis and digitizing tablet. A WACOM Cintiq 21UX digitizing tablet was used to record the handwriting movements of participants. The digitizing tablet has an active display area of 17" by 12.75" and spatial resolution of 5080 lines per inch. Because this tablet provides real-time on-screen visual feedback, a special non-

inking pen was used by participants. MovAlyzeR software (NeuroScript, LLC; Tempe, AZ, USA) was utilized to quantify handwriting movements with a maximum sampling rate of 200 Hz and x-y coordinates were low-pass filtered at 12 Hz. Handwriting movements were broken down by MovAlyzeR software into strokes using interpolated vertical velocity zero crossings. In this sense, a stroke, representing a "unit" of handwriting, can be defined as "a segment bounded by time moments at which the vertical component of the velocity changes sign" (Teulings, Thomassen, & van Galen, 1983, p. 168).

Kinematic variables derived using MovAlyzeR software include Relative intra-individual standard deviation of Peak Velocity (RPV) and Normalized Jerk (NJ). The RPV variable is a coefficient of variation (CV) that was derived by dividing the absolute standard deviation of mean peak velocity of each digitized word or symbol by the average peak velocity of the digitized word or symbol (Mergl et al., 1999). The word "hello" and the novel symbol "  " are described below (see Figure B1 in Appendix B for a scaled version of the novel symbol). The RPV variable reflects stability, coordination, and consistency of an individual's handwriting, with less consistently controlled movements indicated by higher values and more consistently controlled movements reflected by lower values (Mergl et al., 1999; Schroter et al., 2003). An RPV value of 0 would indicate completely identical mean peak velocity across all trials of a writing task.

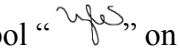
NJ is a measure of writing smoothness and fluency. High NJ scores indicate dysfluent movement and low NJ scores indicate smoother, fluent, and more automatized movement (Teulings et al., 1997; Yan et al., 2008). Said another way, as one practices

and in turn automatizes a graphomotor program, dysfluency decreases (Portier & van Galen, 1992), as will the NJ variable. In turn, the NJ measure should indicate greater dysfluency when individuals write a novel symbol or grapheme on the digitizing tablet versus a well-practiced and automatized symbol or grapheme. The NJ variable is similar to the dysfluency measure of "number of inversions of acceleration" used in much of the research utilizing kinematics to investigate graphomotor problems in those diagnosed with ADHD (for examples, see Flapper et al., 2006; Schoemaker et al., 2005; Tucha & Lange, 2001, 2004, 2005; Tucha et al., 2006; and Tucha et al., 2003) in that NJ "is the change of acceleration per time" (Teulings et al., 1997, p. 160). NJ, however, has the advantage of allowing the comparison of words or symbols of varying size and movement durations because it is normalized (Teulings et al., 1997).

All demographic and research data were kept confidential and secure. Additionally, participant demographic and research data were de-identified (i.e., coded with a randomly assigned participant identification number) but still attached to identifying information for two weeks after the data were collected, thus giving participants the opportunity to withdraw their data from the study. After this time, the link connecting identifying personal information with demographic and research data was removed and only arbitrary participant identification numbers were associated with demographic and research data.

Procedures

In the following order, participants: 1) took part in an interview with the researcher to provide demographic and medical information, 2) answered questions related to ADHD symptomatology, 3) participated in an abbreviated test of general

intellectual ability, 4) signed their name on the digitizing tablet 10 times, 5) wrote the word “hello” in lower-case using cursive handwriting on the digitizing tablet 30 times (representing the automatized condition), and 6) wrote the novel symbol “  ” on the digitizing tablet 30 times (representing the novel word condition). A sample of this word and symbol was visible to the participant on a card throughout the graphomotor task. Instructions for all tasks were given aurally, with instruction provided visually on the digitizing tablet throughout.

All data from control participants was collected in one session. Data obtained from clinical participants was collected on two occasions, once while the participants were taking prescribed dosages of ADHD medication and a second time after abstaining from prescribed dosages of ADHD medication for a 24 to 48 hour period (withdrawal of medication time-frame based on product information indicating extremely low mean drug plasma concentrations between 24 and 48 hours after taking stimulant medication; U.S. Food and Drug Administration, 2007). The time-frame between test and retest for this group was approximately one week (M = 6.75, SD = 0.71). The demographics questionnaire, BAARS-IV, and WAIS-IV subtests were completed while the clinical participants were taking prescribed dosages of ADHD medication to minimize potential discomfort associated with the return of ADHD symptomatology combined with a relatively long research process. The Current Symptoms form of the BAARS-IV questionnaire was completed while clinical participants were off of their prescribed ADHD medication. Experimental task administration within the context of medication status was counterbalanced so that half of the clinical participants completed the writing

tasks while taking prescribed medication on their first visit, while the other half completed the writing tasks while taking prescribed medication during their second visit.

To become familiarized with using the digitizing tablet and pen, all participants began the writing task by signing their name on the digitizing tablet 10 times. Subsequently, participants began writing experimental trials. No specific instructions were given related to the quality of the handwriting participants were to produce beyond pointing to the sample and telling the participants to "Write the word hello in cursive and lower case as it is written on the card. Just write how you typically write." When the researcher was pressed further for additional instruction, participants were only told to "Simply write how you typically write in cursive." In the case of the novel symbol, participants were instructed as follows: "Here is another symbol for the word 'hello.' Please write the symbol as demonstrated on the card." If participants questioned whether neatness was required, the investigator stated, "Just write it how you would write any other word, but make it look like the symbol as demonstrated on the card." Because handwriting is variable within individuals, even when writing the same grapheme, participants wrote each word and symbol 30 times in order to acquire a statistically stable sample of handwriting. Finally, all participants were given the ability to manipulate the position of the tablet to one that was comfortable for writing, as well as position the cards containing the word "hello" and the novel symbol wherever was best for them.

CHAPTER IV

ANALYSIS OF RESULTS

All data analyses were performed using IBM SPSS Statistics, Version 20.0. Unless otherwise noted, an alpha level of .05 was used to determine statistical significance. In addition, interpretations of effect sizes using ω^2 were based on Kirk's (2003) guidelines, such that 0.010 was interpreted as a small association, 0.059 as a medium association, and 0.138 or larger as a large association.

Data Analysis of Assumptions

Prior to hypothesis testing, the data were analyzed to determine adherence to the assumptions of ANOVA, repeated measures ANOVA, and mixed design ANOVA. Cumulatively, tested assumptions included normality of distribution and homogeneity of variance. The assumptions of sphericity and homogeneity of variance/covariance matrices by group were not analyzed due to the research design only incorporating two levels of repeated measures. Assumptions were analyzed using the variables Estimated IQ, Current Total ADHD Score, Childhood Total ADHD Score, NJ under the automatized writing task condition, and RPV under both the automatized and novel writing task conditions, with group membership (i.e., control versus clinical participants) as the independent variable (IV).

Homogeneity of variance. Homogeneity of variance was assessed by first identifying outlier variables (i.e., data with derived *z*-scores greater than |2.5|) and next using Levene's test of equality of error variances, with statistical significance of the latter (i.e., $p < .05$) reflecting a potential violation of this assumption. The following outliers were identified: one control participant within the Estimated IQ dependent variable (DV)

and one clinical participant within both the NJ DV during automatized graphomotor execution while on ADHD medication as well as within the NJ DV during automatized graphomotor execution while off ADHD medication. In turn, all subsequent analyses of assumptions were conducted with and without the inclusion of outliers for comparative purposes. A significant Levene's statistic was found between the variances of the ADHD group on medication and control participants within the NJ DV during the automatized writing task. No other statistical significance was found using Levene's test, indicating that the variation within conditions was roughly equivalent for all other comparisons. When outliers were removed from the dataset, homogeneity of variance statistics improved for the NJ DV during the automatized writing task when comparing control participants versus clinical participants on ADHD medication, but statistical significance persisted. Removing the outlier found within the Estimated IQ DV did not affect the non-significant finding of Levene's test of homogeneity of variance, and in fact moved the data closer towards heterogeneity of variance.

It is important to note that ANOVA may be robust to violations of homogeneity of variance when comparison groups are equal or nearly equal in size (i.e., the larger group contains less than 1.50 times the number of participants than the smaller group) and when the variance distribution between the largest and smallest variances is not greater than a 4:1 ratio. The control group was 3.75 times larger than the clinical group and 4.29 times larger than the clinical group for comparisons in which outliers were removed. In addition, when outliers were retained, the variance distribution between the largest and smallest variance in NJ data during the automatized writing task comparing the ADHD group on medication and control participants was over 20:1. When outlier

data was removed, the variance ratio in this comparison decreased substantially to almost 5:1. Variance data are presented in Table 2 and the results of homogeneity of variance testing are summarized in Table 3.

Table 2

Variance of Data Within Conditions

Dependent Variable	Group	Variance (With Outliers)	Variance (Without Outliers)
Estimated IQ	Control	122.01	88.92†
	ADHD	201.84	201.84
Current ADHD	Control	63.10	63.10
	ADHD	117.64	117.64
Childhood ADHD	Control	60.44	60.44
	ADHD	54.12	54.12
NJ – Auto	Control	50.66	50.66
	On Rx	1028.50	243.96†
	Off Rx	715.81	98.98†
RPV – Auto	Control	0.0015	0.0015
	Off Rx	0.0019	0.0019
RPV – Novel	Control	0.0026	0.0026
	Off Rx	0.0037	0.0037

Note. IQ = Estimated Full Scale IQ; NJ = Normalized Jerk; RPV = Relative Intraindividual Peak Velocity. Rx = Clinical/ADHD participants' medication status (On or Off medication). † = a change in value from "with outliers" to "without outliers."

Table 3

Levene's Test of Homogeneity of Variance

Dependent Variable	Group Comparison	*F*		*df*		Sig.	
		w	w/o	w	w/o	w	w/o
Estimated IQ	Control vs. ADHD	0.58	1.56	1, 36	1, 35	.45	.22
Current ADHD	Control vs. ADHD	0.14	0.14	1, 36	1, 36	.71	.71
Childhood ADHD	Control vs. ADHD	0.04	0.04	1, 36	1, 36	.85	.85
NJ – Auto	Control vs. On Rx	22.50	10.02†	1, 36	1, 35	.00*	.00*
NJ – Auto	Control vs. Off Rx	12.40	2.75†	1, 36	1, 35	.70	.08
NJ – Auto	On Rx vs. Off Rx	0.30	1.25†	1, 14	1, 12	.59	.29
RPV – Auto	Control vs. Off Rx	0.22	0.22	1, 36	1, 36	.64	.64
RPV – Novel	Control vs. Off Rx	0.17	0.17	1, 36	1, 36	.68	.68

Note. Analyses of the assumption of homogeneity of variance within the data. IQ = Estimated Full Scale IQ; NJ = Normalized Jerk, RPV = Relative Intraindividual Peak Velocity; Auto = automatized writing condition; Rx = ADHD participants' medication status (On or Off medication); w = results with outliers; w/o = results without outliers. * = statistical significance ($p < .05$) and violation of the assumption of homogeneity of variance. † = a change in value from "with outliers" to "without outliers."

Normality and Independence of Observations. The assumption of normality was tested by analyzing skewness, kurtosis, and Shapiro-Wilk statistics. With the inclusion of outlier scores, skewness *z*-scores were outside conventional cut-offs of significance (i.e., skewness greater than |2|) for the NJ DV within the control group under the automatized condition as well as for the NJ DV within the clinical group off ADHD medication, both of which indicated a positive skew and a potential violation of the assumption of normality. All other cells did not reflect significant positive or negative skewness. Kurtosis *z*-scores, however, were greater than conventional cutoffs (i.e.,

kurtosis greater than |3|) for the NJ DV under the automatized writing task for controls, clinical participants on ADHD medication, and clinical participants off ADHD medication, indicating significant leptokurtic kurtosis. The Shapiro-Wilk statistic was also statistically significant for these same cells as well as for the Current Total ADHD Score as measured by the BAARS-IV. Within the RPV DV, no significant skewness or kurtosis was observed, and the Shapiro-Wilk statistic was non-significant for all data cells for this variable. This in turn indicated that RPV results represented the only normally distributed experimental data. Removing outlier data resulted in normalizing the distribution of data with regard to skewness and kurtosis for all data cells with the exception of the NJ DV under the automatized writing task within the control group, which retained its significantly positive skew and significantly leptokurtic distribution. In addition, the NJ DV under the automatized writing task continued to produce a significant Shapiro-Wilk statistic within the clinical participant group on ADHD medication.

ANOVA is said to be robust to violations of normality when sample sizes are large and group sizes are roughly equivalent. Noting the relatively small sample size and large group size differences within the sample, the violations of normality found within the NJ DV while retaining outlier variables would significantly impact the reliability of the ANOVA *F* statistic for all comparisons involving the NJ DV. Removing outlier data normalized the distribution of NJ results for clinical participants off ADHD medication, but not for NJ results for the control group or clinical participants on ADHD medication. Tests of normality data, with and without outliers, are summarized in Table 4.

Table 4

Normality of Data

Dependent Variable	Group	Skewness w	Skewness w/o	Kurtosis w	Kurtosis w/o	Shapiro-Wilk w	Shapiro-Wilk w/o
Estimated IQ	Control	0.58	-0.14†	1.03	-0.70†	.34	.32†
	Clinical	-0.52	-0.52	-0.12	-0.12	.78	.78
Current Total ADHD Score	Control	1.10	1.10	1.58	1.58	.03*	.03*
	Clinical	0.30	0.30	-1.72	-1.72	.20	.20
Childhood Total ADHD Score	Control	0.02	0.02	-1.14	-1.14	.23	.23
	Clinical	-0.40	-0.40	-1.14	-1.14	.31	.31
NJ – Auto	Control	2.28*	2.28*	7.63*	7.63*	.00*	.00*
	On Rx	1.89	1.40†	3.53*	0.73†	.01*	.03*†
	Off Rx	2.19*	1.11†	5.09*	0.44†	.00*	.17†
RPV - Auto	Control	0.02	0.02	0.10	0.10	.31	.31
	Off Rx	1.38	1.38	1.88	1.88	.15	.15
RPV - Novel	Control	0.42	0.42	-0.89	-0.89	.06	.06
	Off Rx	0.93	0.93	-0.86	-0.86	.06	.06

Note. Analyses of the assumption of normality of distribution within the data. NJ = Normalized Jerk; RPV = Relative Intraindividual Peak Velocity; Auto = Automatized writing task; Rx = ADHD participants' medication status (On or Off medication); w = results with outliers; w/o = results without outliers. * = statistical significance ($p < .05$) and violation of the assumption of normality. † = a change in value from "with outliers" to "without outliers."

Finally, data were gathered from participants in individual sessions. Combined with the general novelty of the experimental tasks utilized, lack of known organized communication between participants, and the manner in which data were gathered, it is unlikely that participants' scores were systematically related.

Taken together, the non-normally distributed data on the NJ DV for control participants and clinical participants on ADHD medication combined with the

heterogeneity of variance found comparing these two groups increases the risk of Type 1 errors when conducting analyses based on NJ data. In addition, largely unequal sample sizes and variance distributions greater than 4:1 make conclusions drawn from NJ DV results during the automatized writing task tenuous due the additional violation in the assumption of normality. Again, removing outlier data improved the normality of the data, but did not eliminate non-normality entirely. Given the above stated issues associated with the data and the assumptions of ANOVA, 1) all subsequent analyses were conducted without the presence of outlier scores on the NJ DV and 2) nonparametric statistical analyses were also conducted for comparisons between control and clinical participants on ADHD medication that involve using the NJ DV in order to provide support for, or against, significant findings that were found using parametric statistics.

Demographics and ADHD Symptomatology

Data pertaining to participant demographics, estimated IQ, and presence of ADHD symptomatology were collected for the purposes of sample description. ANOVAs were performed to determine significant initial group differences when appropriate.

In the overall sample, more participants were right-handed (84.21%) than left-handed (15.79%), more women (78.95%) participated in the study than men (21.05%), and a majority of the participants self-identified as Caucasian (71.05%). Between the control and clinical participant groups, a greater proportion of clinical participants were left-handed (37.50%) than control participants (10.00%). Fortunately, kinematic variables have not been shown to be affected by handedness alone (Mergl et al., 1999). There was a greater proportion of men in the clinical participant sample (62.50%) than in

the control participant sample (10.00%). A slightly greater representation of non-dominant ethnic/racial group members was also observed in the clinical participant sample (33.33%) versus the control participant sample (20.00%).

Overall, control participants (M = 27.56, SD = 11.91) were younger than clinical (M = 35.00, SD = 9.08) participants, but not significantly, $F(1, 36) = 2.69$, $p = .110$, and with a small effect size, $\omega^2 = .043$. There was, however, a broader age range in control participants (age range: 18.58–54.08 years) versus clinical participants (age range: 23.25–46.60 years). Control (M = 94.17, SD = 11.05) and ADHD (M = 94.88, SD = 14.21) participants performed nearly identically on the general test of intellectual ability as a group, $F(1, 36) = 0.02$, $p = .880$, $\omega^2 = .000$, with both groups falling within the average range. Please see Table 5 for a summary of all participant demographic information.

Table 5

Participant Demographic Information

	Control			ADHD		
	n	*M*	*SD*	*n*	*M*	*SD*
Handedness						
Right	27	-	-	5	-	-
Left	3	-	-	3	-	-
Sex						
Women	27	-	-	3	-	-
Men	3	-	-	5	-	-
Race/Ethnicity						
Asian	2	-	-	0	-	-
Black	3	-	-	1	-	-
Caucasian	24	-	-	6	-	-
Hispanic	1	-	-	1	-	-
Age	-	27.56	11.91	-	35.00	9.08
Estimated IQ	-	94.17	11.05	-	94.88	14.21

Note. Estimated IQ = estimate of general intellectual ability. ADHD = clinical participants diagnosed with ADHD.

Participant ratings of ADHD symptomatology as measured by the BAARS-IV are summarized in Table 6. Clinical participants rated current ADHD symptomatology (M = 48.88, SD = 7.86) as occurring significantly more frequently than control participants (M = 30.00, SD 7.94), $F(1, 36) = 35.80$, $p < .001$, with an observed large effect size, ω^2 = .478. In addition, clinical participants reported significantly more symptoms of ADHD that occurred during childhood (M = 50.88, SD = 7.36) than did control participants (M = 30.67, SD = 7.77), $F(1, 36) = 43.56$, $p < .001$, also with a large effect size, $\omega^2 = .528$.

Table 6

BAARS-IV ADHD Symptomatology

	Control		Clinical	
	M	*SD*	*M*	*SD*
Current Symptom Total ADHD Score:	30.00	7.94	48.88	7.86
Childhood Symptom Total ADHD Score:	30.67	7.77	50.88	7.36

As shown in Table 7, a greater proportion of clinical participants also reported clinically significant levels of current (100%) and childhood (87.50%) ADHD symptomatology compared to control participants (significant levels of current ADHD symptomatology = 13.3%, significant levels of childhood ADHD symptomatology = 0%). Taken together, it can be reasonably concluded that clinical participants reported significantly higher levels of current and childhood ADHD symptoms than control participants. Finally, ADHD subtypes diagnosed in clinical participants included ADHD-C (n = 5), ADHD-PI (n = 1), and ADHD-HI (n = 2).

Table 7

Number of Participants with BAARS-IV Scores ≥ 93rd %tile

	Control	Clinical
	n	n
Current Symptom Total ADHD Score:	4 (13.30%)	8 (100%)
Childhood Symptom Total ADHD Score:	0 (0.00%)	7 (87.50%)

Kinematic Analyses

Overlap with past research. Due to multiple comparisons and the relatively low risk involved with rejecting a true null hypothesis, a Bonferroni correction was used for all statistical comparisons. As such, the alpha level for statistical comparisons made to determine support for past research was adjusted to .02. Two One-Way ANOVAs were used to compare the handwriting fluency of controls with clinical participants both on ADHD medication as well as off ADHD medication. Between group comparisons based on fluency measures detected no statistically significant differences between control (M = 18.43, SD = 7.12) and clinical participants taking ADHD medication (M = 25.57, SD = 15.62), $F(1, 35) = 3.45$, $p = .072$, but did demonstrate a medium effect size, $\omega^2 = .062$. This non-significant finding was consistent with previous research. Nonparametric statistical analysis (i.e., the Mann-Whitney U Test) also indicated that this difference was not statistically significant, $p = .435$, and that the null hypothesis should be retained. The handwriting fluency scores of clinical participants off ADHD medication (M = 21.10, SD = 10.47) compared to those of control participants (M = 18.43, SD = 7.12) were not significantly different, $F(1, 35) = 0.66$, $p = .421$, and demonstrated an uninterpretable effect size, $\omega^2 = .000$. Combined, these non-significant findings are consistent with previous research.

A repeated measures One-Way ANOVA was used to examine automatized handwriting fluency (i.e., NJ) of clinical participants taking ADHD medication versus those same participants discontinuing their ADHD medication for 24 hours. The results comparing the handwriting fluency of clinical participants on ADHD medication (M = 25.57, SD = 15.62) versus off ADHD medication (M = 21.10, SD = 10.47) did not

identify a statistically significant difference, $F(1, 6) = 1.51$, $p = .265$. This result is consistent with previous findings in that handwriting fluency of clinical participants did not differ significantly based on medication status. Although not a statistically significant difference, a medium effect size was found, $\omega^2_{partial} = .068$. See Table 8 for a summary of automatized fluency results as well as source data for statistical comparisons and Figure 3 for a graphical comparison of the automatized writing fluency of control participants versus clinical participants both on and off ADHD medication.

Table 8

Automatized Writing Fluency – Summary and Source Table

Source	*M*	*SD*	*SS*	*df*	*MS*	*F*	*p*	ω^2
Group Membership	-	-	289.14	1	289.14	3.45	.072	.062
Error $_{Between}$	-	-	2932.99	35	83.80	-	-	-
Control	18.43	7.12	-	-	-	-	-	-
On Rx	25.57	15.62	-	-	-	-	-	-
Group Membership	-	-	40.24	1	40.24	0.66	.421	.000
Error $_{Between}$	-	-	2127.38	35	60.78	-	-	-
Control	18.43	7.12	-	-	-	-	-	-
Off Rx	21.01	10.47	-	-	-	-	-	-
Medication Status	-	-	70.09	1	70.09	1.51	.265	.068†
Error $_{Residual}$			278.44	6	46.41	-	-	-
On Rx	25.57	15.62	-	-	-	-	-	-
Off Rx	21.10	10.47	-	-	-	-	-	-

Note. On Rx = ADHD/Clinical participants on ADHD medication; Off Rx = ADHD/Clinical participants off ADHD medication. M = mean of normalized jerk (NJ) value; SD = standard deviation of normalized jerk (NJ) value. † = partial omega-squared.

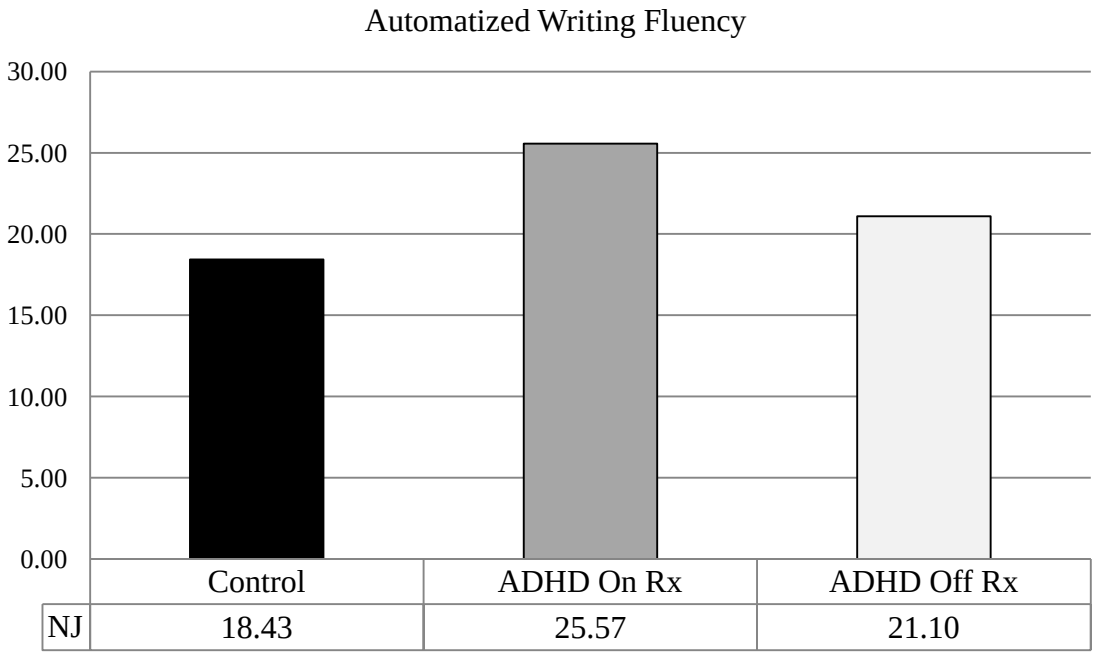


Figure 3. Automatized writing fluency of control and clinical participants. NJ = Normalized Jerk. Higher values of NJ indicate more dysfluent writing, whereas lower values of NJ indicate more fluent and automatized writing. ADHD On Rx = ADHD/Clinical participants on ADHD medication; ADHD Off Rx = ADHD/Clinical participants off ADHD medication.

Variability of graphomotor performance. The alpha level was set at .05 to indicate statistical significance for the following comparisons. A One-Way ANOVA was used to analyze the kinematic variability of automatized handwriting performance (i.e., RPV) of healthy control participants with that of ADHD participants that discontinued ADHD medication for 24 hours. No main effect was found related to group membership and variability of graphomotor performance, $F(1, 36) = 0.37$, $p = .545$, and no interpretable effect size was found, $\omega^2 = .000$, indicating that clinical participants not taking ADHD medication ($M = 0.12$, $SD = 0.06$) demonstrated similar variability in automatized graphomotor performance to control participants ($M = 0.13$, $SD = 0.04$). See Table 9 for a summary of graphomotor variability findings as well as source information. See Figure 4 for a graphical comparison of graphomotor variability findings.

Table 9

Automatized Writing Variability – Summary and Source Table

Source	*M*	*SD*	*SS*	*df*	*MS*	*F*	*p*	ω^2
Group Membership	-	-	.001	1	.001	0.37	.545	.000
Error $_{Between}$	-	-	.057	36	.002	-	-	-
Control	.13	.04	-	-	-	-	-	-
Off Rx	.12	.04	-	-	-	-	-	-

Note. Off Rx = ADHD/Clinical participants off ADHD medication. M = mean of the relative intraindividual mean peak velocity (RPV) value; SD = standard deviation of the relative intraindividual mean peak velocity (RPV) value.

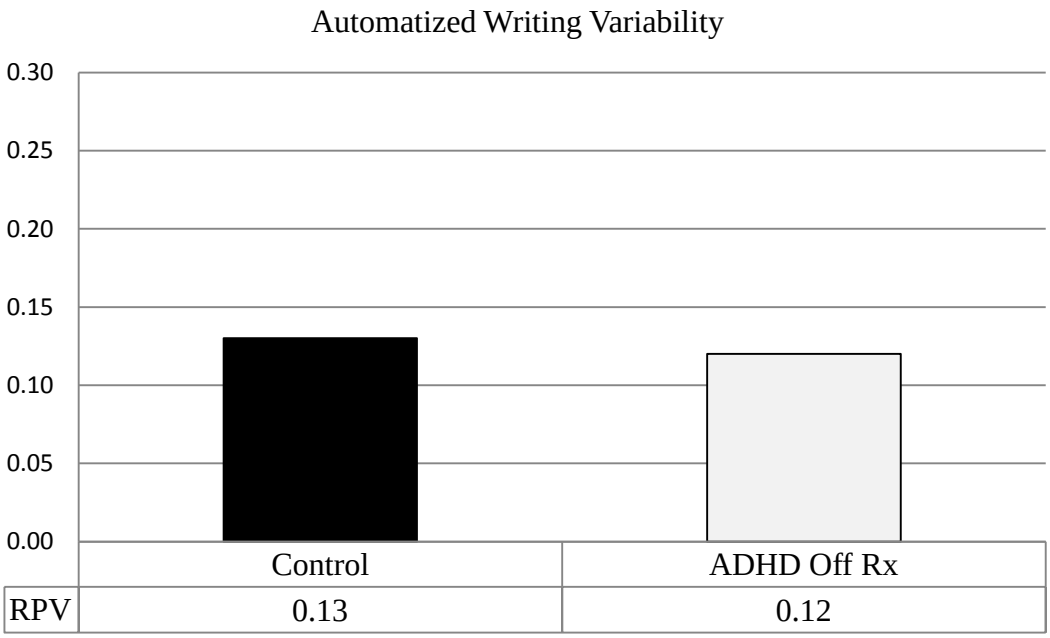


Figure 4. Variability of handwriting performance in control and clinical participants. RPV = Relative Intraindividual Mean Peak Velocity. Less consistently controlled movements are indicated by higher values and more consistently controlled movements reflected by lower values. ADHD Off Rx = ADHD/Clinical participants off ADHD medication.

The effects of novelty on variability. A 2 x 2 factorial mixed design ANOVA was used to compare the effects of novelty on variability measures in those diagnosed with ADHD off medication versus healthy controls. There was a significant main effect

for writing task, $F(1, 36) = 24.86$, $p < .001$, indicating that overall, more variable and less consistent handwriting was seen in participants when performing the novel writing task ($M = 0.18$, SD 0.06) versus the automatized writing task ($M = 0.12$, $SD = 0.04$). The effect size for the writing task main effect was large, $\omega^2_{partial} = .333$. However, no significant interaction effect was observed, $F(1, 36) = 0.11$, $p = .740$, with no interpretable effect size, $\omega^2_{partial} = .000$. See Table 10 for source information pertaining to graphomotor variability findings as a function of writing task. See Figure 5 for a graphical comparison of graphomotor variability findings related to writing task.

Table 10

Graphomotor Variability as a Function of Novelty –Source Table

Source	*SS*	*df*	*MS*	*F*	*p*	ω^2
Within Subjects						
Writing Task	0.040003	1	0.040003	24.86	<.001*	.333
Error $_{Within}$	0.057937	32	0.001609	-	-	-
Interaction						
Writing Task x Group Membership	0.000180	1	0.000180	0.11	.740	.000
Error $_{Interaction}$	0.057937	32	0.001636	-	-	-

Note. * = statistical significance $p < .05$

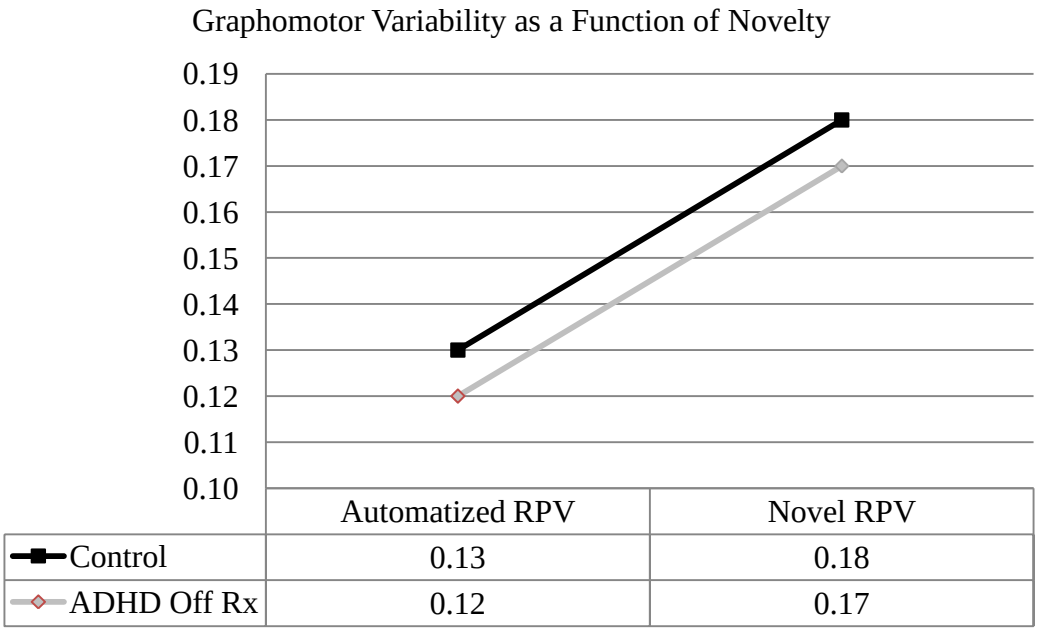


Figure 5. Handwriting variability based on writing task. RPV = Relative Intraindividual Mean Peak Velocity. Less consistently controlled movements are indicated by higher values and more consistently controlled movements reflected by lower values. ADHD Off Rx = ADHD/Clinical participants off ADHD medication.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Discussion

Utilizing a digitizing tablet and specialized kinematic analyses software to quantify graphomotor performance during putatively automatized and novel writing tasks in adults with and without a diagnosis of ADHD, the present study sought to: 1) determine whether the variability of performance observed in other psychological domains in those diagnosed with ADHD would manifest within kinematic variables associated with stability and coordination of graphomotor output (i.e., RPV) and 2) determine whether a novel writing task would differentially affect the variability of graphomotor output of adults diagnosed with ADHD compared to healthy controls.

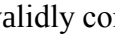
Overlap with past research. Consistent with prior research (Tucha & Lange, 2004), results of the present study suggest that automatized graphomotor fluency, as measured by kinematic analysis, is not significantly different in adults diagnosed with ADHD taking prescribed dosages of stimulant medication from that of neurotypical adults. This conclusion was supported using both parametric and nonparametric statistical analysis. Despite these non-statistically significant findings that appear to corroborate past research, this conclusion should be accepted cautiously. Findings of statistical significance using ANOVA or other analyses under the general linear model are affected by sample size. That is, as sample size increases, the likelihood of finding a statistically significant result continues to increase, even when differences in performance are relatively small. As such, the power of the research design, which takes into account sample size, effect size, and alpha level, must also be considered. Noting the medium

effect size (ω^2 = .062) and the relatively small number of clinical participants (n = 7) within this study, it is likely that this non-statistically significant finding is due to low power rather than non-significant differences between healthy adults and adults diagnosed with ADHD on stimulant medication. In turn, a statistically significant result may have been found if the clinical sample was larger and fluency results within the study maintained the same pattern.

Also consistent with previous research, adults diagnosed with ADHD off medication produced similarly fluent automatized graphomotor output as control participants without ADHD. Although an argument could be made that increased power via a larger sample of clinical participants may result in the formulation of a different conclusion, the extremely small (and uninterpretable) effect size observed in this study combined with both the adherence to the assumptions of ANOVA and findings that are consistent with previous research strongly suggest that this conclusion is reliable and valid.

Concerning the handwriting fluency of adults diagnosed with ADHD on stimulant medication versus those same adults off ADHD medication, the present study was consistent with previous research indicating no statistically significant difference in automatized graphomotor fluency. This finding suggests that medication status may not affect the graphomotor fluency of adults diagnosed with ADHD when performing an automatized writing task. Again, this interpretation must be made with caution. Similar to the above results comparing the automatized graphomotor fluency of control participants with that of clinical participants on ADHD medication, the medium effect size (ω^2 = .068) found in the comparison of adults with ADHD on medication versus off

medication suggests that non-significant findings could be the result of insufficient statistical power associated with a small clinical participant sample.

However, if statistical power were sufficient and statistically significant findings related to differences in automatized graphomotor fluency were found between adults with ADHD on stimulant medication versus off ADHD medication, as well as between adults with ADHD on stimulant medication versus control participants, results would still need to be interpreted cautiously due to other methodological considerations. First, graphomotor fluency in previous research was operationalized as the mean number of inversions in the direction of vertical velocity over time (Tucha & Lange, 2004). In this study, however, graphomotor fluency was operationalized as normalized jerk, which is derived from the number of changes in acceleration in time (analogous to the number of changes in velocity over time) but was then normalized due to the effects of size and duration of movements on fluency measures (Tuelings et al., 1997). Using the NJ variable as opposed to the mean number of inversions in velocity provided the benefit of validly comparing graphomotor fluency of the word "hello" and the symbol "  ." However, the NJ variable may not be completely analogous to previously used measures quantifying automatized graphomotor fluency in adults with ADHD. As such, the derived fluency measures of this study may not be completely comparable with those of past research investigating graphomotor fluency in adults with ADHD.

Secondly, the previous study investigating graphomotor fluency in adults diagnosed with ADHD analyzed the specific letter combination of "ll" within two German words (Tucha & Lange, 2004). The present study, however, analyzed the entire word "hello" to determine automatized graphomotor fluency. Intuitively, the

biomechanical forces necessary to generate the letters “ll” versus the word “hello” are different based upon salient graphemic differences. In turn, conclusions drawn from comparisons between the current study and past research should be done with caution noting that differences in graphomotor fluency findings may be due, at least in part, to the experimental stimulus used.

Variability of graphomotor performance. Using the kinematic variable RPV, clinical participants exhibited variability in graphomotor output when executing an automatized writing task that was similar to that of control participants. As such, the results of this study suggest that the variability of performance observed in various psychological domains (e.g., emotional expression, handwriting production, fine motor skill movements, motor coordination, and motor force output) within the ADHD population may not be manifest within the kinematic measures of stability, consistency, and coordination used in this study, in which participants performed an automatized graphomotor task.

This was the first such research to utilize RPV as a measure of variability of graphomotor performance in adults diagnosed with ADHD. Previous research utilizing the RPV measure to study kinematic aspects of handwriting in clinical populations focused on patients diagnosed with probable Alzheimer’s dementia (AD) (Schroter et al., 2003). In this research, Schroter and colleagues found that when performing a spiral drawing task, participants with probable AD exhibited significantly more variability, incoordination, and greater inconsistency in the kinematics of handwriting movements compared to similarly aged healthy control participants. One of the primary rationales for conducting this study was the high co-occurrence of salient motor dysfunction found

within the AD population: a high prevalence of Parkinsonism and various extrapyramidal motor symptoms. Although motor problems have also been documented in those with ADHD, motor sequelae in the ADHD population may be more subtle than what is observed in patients with AD. Accordingly, a failure to demonstrate significant differences in consistency of graphomotor output between adults with ADHD and healthy controls could be the result of less than optimal sensitivity of the RPV measure in detecting subtle motor differences in the ADHD population.

Further, how the RPV measure was derived may have created an insensitive measure that failed to detect intraindividual variability of graphomotor production. That is, the mean peak velocity was collapsed across all strokes within each trial and then averaged with all 30 trials of each writing task. The RPV, which is a coefficient of variation (CV), was then determined for each participant by dividing the standard deviation of the mean peak velocities by the average of the mean peak velocities across all trials. As such, this collapsed mean may not have optimally reflected variability of graphomotor performance with sufficient sensitivity because variability within each individual writing trial was not taken into consideration when calculating the RPV. Said another way, deriving the RPV variable to indicate intraindividual variability by creating a CV based on the averages and standard deviation of mean peak velocities across all trials rather than deriving the RPV variable based upon an average of the coefficients of variation of mean peak velocities calculated within each trial may have underestimated the intraindividual variability of automatized and novel graphomotor output.

The writing tasks themselves may also not have optimally allowed participants to demonstrate significant variability of graphomotor performance. For example, previous

work utilizing the RPV variable has demonstrated no statistically significant differences in intraindividual variability of handwriting between men and women when writing letters or simple geometric figures (Mergl et al., 1999). However, in this same study, statistically significant gender differences in intraindividual variability of graphomotor performance emerged when participants executed a six word sentence.

Bearing in mind the methodological and statistical considerations mentioned above, the findings of the present study suggest the following: 1a) conclusions concerning the fluency of automatized graphomotor performance in healthy controls as compared with adults diagnosed with ADHD taking stimulant medication can only be tentatively drawn at this time. These findings, in combination with past findings, suggest that the automatized graphomotor fluency between these groups is similar. However, medium effect sizes suggest that real differences may be present and would be detected if statistical power were greater. 1b) A negligible observed effect size, non-significant findings, and replication of past research provide strong evidence that the graphomotor fluency of adults with ADHD off stimulant medication is similar to the fluency of individuals without ADHD. 2) Conclusions regarding the fluency of automatized graphomotor performance in adults with ADHD on stimulant medication versus off ADHD medication can only be tentatively drawn. The medium effect size found within this comparison suggests potential differences in automatized graphomotor fluency in adults with ADHD on stimulant medication versus off medication. 3) Variability of performance, as measured by the RPV variable, does not appear to manifest within the graphomotor domain in adults diagnosed with ADHD. However, more sensitive measures of intraindividual variability of graphomotor performance or different writing

tasks may yield different results and in turn, support an alternative conclusion. 4) The novelty of a graphomotor task does not appear to differentially affect the variability of kinematic handwriting performance in adults diagnosed with ADHD as measured by the RPV variable. As mentioned previously concerning the RPV variable, however, different results may be found by calculating a more sensitive measure of intraindividual variability of graphomotor performance.

An additional aim of this study was to add to the current literature demonstrating that ADHD is not simply a disorder of childhood, but rather, a condition that involves specific motor differences that persist into adulthood. Although differences in motor functioning as measured by the kinematic analyses utilized in this study were not statistically significant, effect size differences and methodological considerations do not support the conclusion that motor symptoms do not persist into adulthood. Rather, the results of this study support implementing improved methodology and statistical analyses to further explore potential motor skill differences in those with ADHD that may persist into adulthood.

Further, this study aimed to find support for the use of digitizing technology as an objective diagnostic and descriptive tool within the ADHD population, which would in turn enhance the specificity and/or sensitivity of current assessment and diagnostic techniques. Although significant differences in graphomotor function were not observed between adults with ADHD and healthy controls utilizing the proposed kinematic analyses, when considering the methodological and statistical concerns associated with this study, it is likely too early to conclude that kinematic analysis utilizing digitizing

technology would not create an added benefit when attempting to describe graphomotor performance differences in the adult ADHD population versus unaffected adults.

Methodological Limitations. This project began with several pragmatic considerations, including researching and obtaining appropriate hardware and software, developing research protocols, and learning how to interpret the results of kinematic analyses based on digitizing technology. As such, the present study served as a foundation for future kinematic research utilizing digitizing technology in an effort to understand fine motor and graphomotor skill performance of adults and children with ADHD. An additional benefit of this study was to implement a novel protocol and evaluate the feasibility of future research questions. Although this was not a direct goal, the results and implementation of this project did have the benefit of establishing the limitations of kinematic research utilizing digitizing technology. Nevertheless, methodological limitations and limitations of statistical analyses used may have affected the results, and in turn the conclusions of this study.

Beyond the concerns mentioned above, the primary limitation of this study was the small number of clinical participants that were recruited and as such, low statistical power. Post-hoc power analyses using G*Power software (Buchner, Erdfelder, Faul, & Lang, 2009) confirmed the low power of this study, which ranged from $1\text{-}\beta = .19$ to $1\text{-}\beta = .74$ for all statistical analyses. In addition, when sample sizes are small, data are unstable and yield statistics that are unreliable. An additional problem that this small clinical sample size created was a situation in which the two comparison groups were largely unequal in size. This additional problem further decreases the reliability of the *F* statistic and its ability to help draw conclusions. In the future, recruitment strategies should

involve broadening the pool from which clinical participants are drawn by increasing the number of sources from which participants are recruited. However, the present study could also be considered an initial work in progress, with additional recruitment of participants scheduled to occur in the future.

In addition, parametric statistical analyses in this study utilized the general linear model. These statistical analyses used to determine both group differences in the variability of automatized graphomotor performance and the effect of novelty on these measures of variability may not have been sufficiently sensitive in detecting intraindividual differences. For example, King, Harring, Oliveira, and Clark (in press) utilized both a general linear model statistic (i.e., ANOVA) as well as a random coefficient model technique to study intra- and inter-individual variability of motor movements in healthy children and children diagnosed with DCD. In summary, King and colleagues found that the random coefficient model identified intra- and inter-individual differences in task execution that the general linear model analysis did not detect. As such, the use of ANOVA to analyze intraindividual differences in automatized graphomotor performance may not have been the most appropriate or sensitive statistical model for the purposes of this research question.

Future Research

The results and methodology used in this study suggest multiple lines of research that should be explored in the future. First, the present study sought to understand the variability of graphomotor performance using a CV derived from the average of mean peak velocities for each word or symbol across 30 trials. Alternatively, variability could be expressed as a CV of average fluency results in both automatized and novel writing

tasks. That is, variability of graphomotor performance in adults with ADHD could be explored using a derivative of the NJ variable rather than a derivative of mean peak velocity. In addition, and as mentioned above, future research should attempt to understand variability of graphomotor performance by utilizing an alternative calculation of the intraindividual variation of graphomotor functioning. This would involve computing the average of the CVs of mean peak velocity per trial rather than the CV of the average mean peak velocity collapsed across all trials. Finally, future research into intraindividual variability of performance could benefit from using more powerful and elegant statistical analyses. As demonstrated by King et al. (in press), the use of statistical techniques based upon a random coefficient model may have greater power to detect intraindividual differences in motor skill performance than do techniques based upon the general linear model.

APPENDICES

APPENDIX A

Interview Questionnaire

Name: _____

In what month and year were you born? _____

How would you describe your Sex or Gender? _____

What hand do you primarily use to write with? Right Hand Left Hand Ambidextrous

What medications are you currently taking? Please include dosage information.

Medication	**Dosage**	**Medication**	**Dosage**

Some people use terms such as Arabic, Asian, Black, Hispanic, White, or similar terms to describe their ethnicity. What term would you use to describe your ethnicity? _____

Do you currently have a diagnosis of ADHD? If yes, what is that official diagnosis?

_____

Do you have a current diagnosis or diagnoses affecting the central nervous system or peripheral nervous system that would impair your ability to take part in a writing task? An example of central nervous system diagnosis affecting writing ability includes cerebral palsy affecting the arms and/or hands, and an example of peripheral nervous system diagnosis affecting writing ability includes carpal tunnel.

_____

_____

Is there any other information that you feel may affect your participation in this study that you would like me to know?

_____

_____

APPENDIX B

Figure B1. Scaled version of novel symbol.

REFERENCES

Adi-Japha, E., Landau, Y. E., Frenkel, L., Teicher, M., Gross-Tsur, V., & Shalev, R. S. (2007). ADHD and dysgraphia: Underlying mechanisms. *Cortex, 43*, 700-709. doi:10.1016/S0010-9452(08)70499-4

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Arlington, VA: Author.

American Psychiatric Association. (2010). *DSM-5: Options being considered for ADHD*. Retrieved from http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=383#

Amico, F., Stauber, J., Koutsouleris, N., & Frodi, T. (2010). Anterior cingulated cortex gray matter abnormalities in adults with attention deficit hyperactivity disorder: A voxel-based morphometry study. *Psychiatry Research: Neuroimaging, 191*, 31-35. doi:10.1016/j.pscychresns.2010.08.011

Arnsten, A. F. T., Berridge, C. W., & McCracken, J. T. (2009). The neurobiological basis of Attention-Deficit/Hyperactivity Disorder. *Primary Psychiatry, 16*, 47-54.

Banaschewski, T., Becker, K., Scherag, S., Franke, B., & Coghill, D. (2010). Molecular genetics of attention-deficit/hyperactivity disorder: An overview. *European Child & Adolescent Psychiatry, 19*(3), 237-257. doi:10.1007/s00787-010-0090-z

Barkley, R. A. (2006). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed.). New York: Guilford Press.

Barkley, R. A. (2011). Barkley adult ADHD rating scale-IV (BAARS-IV). New York: Guilford Press.

Barkley, R. A., & Fischer, M. (2010). The unique contribution of emotional impulsiveness to impairment in major life activities in hyperactive children as adults. *Journal of The American Academy of Child & Adolescent Psychiatry*, *49*, 503-513. doi:10.1097/00004583-201005000-00011

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2002). The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal Of Abnormal Psychology*, *111*, 279-289. doi:10.1037/0021-843X.111.2.279

Bart, O., Podoly, T., & Bar-Haim, Y (2010). A preliminary study on the effect of methylphenidate on motor performance in children with comorbid DCD and ADHD. *Research in Developmental Disabilities, 31*, 1443-1447. doi:10.1016/j.ridd.2010.06.014

Batty, M. J., Liddle, E. B., Pitiot, A., Toro, R., Groom, M. J., Scerif, G., . . . Hollis, C. (2010). Cortical gray matter in Attention-Deficit/Hyperactivity Disorder: A structural magnetic resonance imaging study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*, 229-238. doi:10.1016/j.jaac.2009.11.008

Bellgrove, M. A., Phillips, J. G., Bradshaw, J. L., Hall, K. A., Presnell, I., & Hecht, H. (1997). Response programming in dementia of the Alzheimer type: A kinematic analysis. *Neuropsychologia*, *35*, 229-240. doi:10.1016/S0028-3932(96)00081-4

Biederman, J. (2005). Attention-Deficit/Hyperactivity Dosorder: A selective overview. *Biological Psychiatry, 57*, 1215-1220. doi:10.1016/j.biopsych.2004.10.020

Biederman, J., Petty, C. R., Clarke, A., Lomedico, A., & Faraone, S. V. (2011). Predictors of persistent ADHD: An 11-year follow-up study. *Journal Of Psychiatric Research, 45,* 150-155. doi:10.1016/j.jpsychires.2010.06.009

Bo, J., Bastien, A. J., Kagerer, F. A., Contreras-Vidal, J. L., & Clark, J. E. (2008). Temporal variability in continuous versus discontinuous drawing for children with Developmental Coordination Disorder. *Neuroscience Letters, 431,* 215-220. doi:10.1016/j.neulet.2007.11.040

Brossard-Racine, M., Majnemer, A., & Shevell, M. I. (2011). Exploring the neural mechanisms that underlie motor difficulties in children with Attention Deficit Hyperactivity Disorder. *Developmental Neurorehabilitation, 14*(2), 101-111. doi: 10.3109/17518423.2010.547545

Brossard-Racine, M., Majnemer, A., Shevell, M., & Snider, L. (2008). Handwriting performance in children with Attention Deficit Hyperactivity Disorder (ADHD). *Journal of Child Neurology, 23,* 399-406. doi: 10.1177/08833073807309244

Brossard-Racine, M., Majnemer, A., Shevell, M., Snider, L., & Belanger, S. A. (2011). Handwriting capacity in children newly diagnosed with Attention Deficit Hyperactivity Disorder. *Research in Developmental Disabilities, 32,* 2927-2934. doi:10.1016/j.ridd.2011.05.010

Brown, A. B., Biederman, J., Valera, E., Makris, N., Doyle, A., Whitfield-Gabrieli, S., . . . Seidman, L. (2011). Relationship of DAT1 and adult ADHD to task –positive and task-negative working memory networks. *Psychiatry Research: Neuroimaging, 193,* 7-16. doi:10.1016/j.pscychresns.2011.01.006

Buchner, A., Erdfelder, E., Faul, F., & Lang, A. (2009). G*Power (Version 3.1.2)[Computer program]. http://www.psycho.uni-duesseldorf.de/aap/projects/gpower/

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., & ... Biederman, J. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the Counting Stroop. *Biological Psychiatry, 45,* 1542-1552. doi:10.1016/S0006-3223(99)00083-9

Castellanos, F., Giedd, J. N., Berquin, P. C., Walter, J. M., Sharp, W., Tran, T., . . . Rapoport, J. L. (2001). Quantitative brain magnetic resonance imaging in girls with attention-deficit/hyperactivity disorder. *Archives Of General Psychiatry, 58,* 289-295. doi:10.1001/archpsyc.58.3.289

Castellanos, F., Giedd, J. N., Marsh, W. L., & Hamburger, S. D. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives Of General Psychiatry, 53,* 607-616.

Chang, S.-H. & Yu, N.-Y. (2010). Characterization of motor control in handwriting difficulties in children with or without developmental coordination disorder. *Developmental Medicine & Child Neurology, 52,* 244-250. doi: 10.1111/j.1469-8749.2009.03478.x

Cubillo, A., Halari, R., Giampietro, V., Taylor, E., & Rubia, K. (2011). Fronto-striatal underactivation during interference inhibition and attention allocation in grown up children with attention deficit/hyperactivity disorder and persistent symptoms. *Psychiatry Research: Neuroimaging, 193,* 17-27. doi:10.1016/j.pscychresns.2010.12.014

Daly, G. G., Hawi, Z. Z., Fitzgerald, M. M., & Gill, M. M. (1999). Mapping susceptibility loci in attention deficit hyperactivity disorder: preferential transmission of parental alleles at DAT1, DBH and DRD5 to affected children. *Molecular Psychiatry*, *4*, 192-196.

Dooijes, E. H. (1983). Analysis of handwriting movements. *Acta Psychologica*, *54*, 9-114. doi:10.1016/0001-6918(83)90026-4

Durston, S., Hulshoff Pol, H. E., Schnack, H. G., Buitelaar, J. K., Steenhuis, M. P., Minderaa, R. B., & ... van Engeland, H. (2004). Magnetic resonance imaging of boys with attention-deficit/hyperactivity disorder and their unaffected siblings. *Journal Of The American Academy Of Child & Adolescent Psychiatry*, *43*, 332-340. doi:10.1097/00004583-200403000-00016

Durston, S., Tottenham, N. T., Thomas, K. M., Davidson, M. C., Eigsti, I., Yang, Y., . . . Casey, B. J. (2003). Differential patterns of striatal activation in young children with and without ADHD. *Biological Psychiatry*, *53*, 871-878. doi:10.1016/S0006-3223(02)01904-2

Eichhorn, T. E., Gasser, T., Mai, N., Marquardt, C., Arnold, G., Schwarz, J, & Oertel, W. H. (1996). Computational analysis of open loop handwriting movements in Parkinson's Disease: A rapid method to detect dopamimetic effects. *Movement Disorders*, *11*, 289-297.

Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., & Sklar, P. (2005). Molecular Genetics of Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, *57*, 1313-1323. doi:10.1016/j.biopsych.2004.11.024

Flapper, B. T., Houwen, S., & Schoemaker, M. M. (2006). Fine motor skills and effects of methylphenidate in children with attention-deficit-hyperactivity disorder and developmental coordination disorder. *Developmental Medicine & Child Neurology, 48,* 165-169. doi:10.1017/S0012162206000375

Fliers, E. A., Franke, B., Lambregts-Rommelse, N. N. J., Altink, M. E., Buschgens, C. J. M., Nijhuis-van der Sangen, M. W. G., . . . Buitelaar, J. K. (2009). Undertreatment of motor problems in children with ADHD. *Child Adolescent Mental Health, 15,* 85-90. doi:10.1111/j.1475-3588.2009.00538.x

Galli, M., Vimercati, S. L., Stella, G., Caiazzo, G., Norveti, F., Onnis, F., . . . Albertini, G. (2011). A new approach for the quantitative evaluation of drawings in children with learning disabilities. *Research in Developmental Disabilities, 32,* 1004-1010. doi:10.1016/j.ridd.2011.01.051

Gangadhar, G., Joseph, D., Srinivasan, A. V., Subramanian, D., Shivakeshaven, R. G., Shobana, N., & Chakravarthy, V. S. (2009). A computational model of Parkinsonian handwriting that highlights the role of the indirect pathway in the basal ganglia. *Human Movement Science, 28,* 602-618. doi:10.1016/j.humov.2009.07.008

Graham, S., & Weintraub, N. (1996). A review of handwriting research: Progress and prospects. *Educational Psychology Review*, 8, 7. Retrieved from EBSCO*host*.

Grootens, K. P., Vermeeren, L., Verkes, R. J., Buitelaar, J. K., Sabbe, B. G. C., van Veelen, N., . . . Hulstijn, W. (2009). Psychomotor planning is deficient in recent-onset schizophrenia. *Schizophrenia Research, 107,* 294-302. doi:10.1016/j.schres.2008.09.032

Guerfali, W. & Plamondon, R. (1997). Effect of variability on letters generation with the vectorial delta-lognormal model. In N. Murshed & F. Bortolozzi (Series, Ed.), *Advances in Document Image Analysis: Vol. 1339. Lecture Notes in Computer Science* (pp. 74-83). doi:10.1007/3-540-63791-5_5

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., & Novey, E. S. (1991). Corpus callosum morphology in attention deficit-hyperactivity disorder: Morphometric analysis of MRI. *Journal Of Learning Disabilities*, *24*, 141-146. doi:10.1177/002221949102400302

Jahn, T., Cohen, R., Hubermann, W., Mohr, F., Kohler, I., Schlenker, R., . . . Schroder, J. (2006). The Brief Motor Scale (BMS) for the assessment of motor soft signs in schizophrenic psychoses and other psychiatric disorders. *Psychiatry Research, 142*(2-3), 177-189. doi:10.1016/j.psychres.2002.12.002

Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry*, *42*, 487-492. doi:10.1111/1469-7610.00742

King, B. R., Harring, J. R., Oliveira, M. A., & Clark, J. E. (in press). Statistically characterizing intra- and inter-individual variability in children with Developmental Coordination Disorder. *Research in Developmental Disabilities*. doi:10.1016/j.ridd.2010.12.043

Kirk, R. E. (2003). The importance of effect magnitude. In S. F. Davis (Ed.). *Handbook of Research methods in Experimental Psychology* (pp. 88-105). Malden, MA: Blackwell Publishing.

Klimkeit, E. I., Sheppard, D. M., Lee, P., & Bradshaw, J. L. (2004). Bimanual Coordination Deficits in Attention Deficit/Hyperactivity Disorder (ADHD). *Journal of Clinical & Experimental Neuropsychology, 26*(8), 999-1010.

Konrad, K. & Eickhoff, S. B. (2010). Is the ADHD brain wired differently? A review on structural and functional connectivity in Attention Deficit Hyperactivity Disorder. *Human Brain Mapping, 31*, 904-916. doi: 10.1002/hbm.21058

Kushki, A., Schwellnus, H., Ilyas, F., & Chau, T. (2011). Changes in kinetics and kinematics of handwriting during a prolonged writing task in children with and without dysgraphia. *Research in Developmental Disabilities, 32*(3), 1058-1064. doi:10.1016/j.ridd.2011.01.026

Lacquaniti, F. (1989). Central presentations of human limb movements as revealed by studies of drawing and handwriting. *Trends in Neurosciences, 12*(8), 287-291. doi:10.1016/0166-2236(89)90008-8

Lerer, R. J., Artner, J., & Lerer, M. (1979). Handwriting deficits in children with minimal brain dysfunction: Effects of methylphenidate (Ritalin) and placebo. *Journal of Learning Disabilities*, 12(7), 26. Retrieved from EBSCO*host*.

Liston, C., Cohen, M. M., Teslovich, T., Levenson, D., & Casey, B. J. (2011). Atypical prefrontal connectivity in Attention-Deficit/Hyperactivity Disorder: Pathway to disease or pathological end point? *Biological Psychiatry, 69*, 1168-1177. doi:10.1016/j.biopsych.2011.03.022

Mackie, S., Shaw, P., Lenroot, R., Pierson, R., Greenstein, D. K., Nugent, T. F., . . . Rapoport, J. L. (2007). Cerebellar development and clinical outcome in Attention

Deficit Hyperactivity Disorder. *The American Journal of Psychiatry*, *164*, 647-655.

Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P., & LaPadula, M. (1998). Adult psychiatric status of hyperactive boys grown up. *The American Journal Of Psychiatry*, *155*, 493-498.

Margolin, D. I. & Wing, A. M. (1983). Agraphia and micrographia: Clinical manifestations of motor programming and performance disorders. *Acta Psychologica*, *54*, 263-283. doi:10.1016/0001-6918(83)90039-2

Martin, N. C., Piek, J. P., & Hay, D. (2006). DCD and ADHD: A genetic study of their shared aetiology. *Human Movement Science*, *25*, 110-124. doi:10.1016/j.humov.2005.10.006

McAlonan, G. M., Cheung, V., Cheung, C., Chua, S. E., Murphy, D. G. M., Suckling, J., . . . Ho, T. P. (2007). Mapping brain structure in attention deficit-hyperactivity disorder: A voxel-based MRI study of regional gray and white matter volume. *Psychiatry Research: Neuroimaging*, *154*, 171-180. doi:10.1016/j.pscychresns.2006.09.006

Mergl, R., Tigges, P., Schroter, A., Moller, H.-J., & Hegerl, U. (1999). Digitized analysis of handwriting and drawing movements in healthy subjects: Methods, results, and perspectives. *Journal of Neuroscience Methods*, *90*, 157-169. doi:10.1016/S0165-0270(99)00080-1

Meulenbroek, R. G. J. & Thomassen, A. J. W. M. (1993). Exploitation of elasticity as a biomechanical property in the production of graphic stroke sequences. *Acta Psychologica*, *82*(1-3), 313-327. doi:10.1016/0001-6918(93)90018-M

Meulenbroek, R. G. J. & van Galen, G. P. (1988). Foreperiod duration and the analysis of motor stages in a line-drawing task. *Acta Psychologica, 69,* 19-34. doi:10.1016/0001-6918(88)90027-3

Meyer, A & Sagvolden, T. (2006). Fine motor skills in South African children with symptoms of ADHD: Influence of subtype, gender, age, and hand dominance. *Behavioral and Brain Functions, 2,* 33. doi:10.1186/1744-9081-2-33

MovAlyzeR [Computer software]. Tempe, AZ: NeuroScript, LLC.

Narr, K. L., Woods, R. P., Lin, J., Kim, J., Phillips, O. R., Del'Homme, M., . . . Levitt, J. G. (2009). Widespread cortical thinning is a robust anatomical marker for Attention Deficit/Hyperactivity Disorder (ADHD). *Journal of The American Academy Of Child & Adolescent Psychiatry, 48,* 1014-1022. doi:10.1097/CHI.0b013e3181b395c0

Overvelde, A. & Hulstijn, W. (2011). Handwriting development in grade 2 and grade 3 primary school children with normal, at risk, and dysgraphic characteristics. *Research in Developmental Disabilities, 32,* 540-548. doi:10.1016/j.ridd.2010.12.027

Overvelde, A. & Hulstijn, W. (in press). Learning new movement patterns: A study on good and poor writers comparing learning conditions emphasizing spatial, timing or abstract characteristics. *Human Movement Science*. doi:10.1016/j.humov.2010.08.016

Pereira, H. S., Eliasson, A.-C., & Forssberg, H. (2000). Detrimental neural control of precision grip lifts in children with ADHD. *Developmental Medicine & Child Neurology, 42,* 545-553.

Phillips, J. G., Bradshaw, J. L., Chiu, E. Teasdale, N., Iansek, R., & Bradshaw, J. A. (1996). Bradykinesia and movement precision in Huntington's disease. *Neuropsychologia, 34,* 1241-1245. doi:10.1016/0028-3932(96)00049-8

Phillips, J. G., Chiu, E., Bradshaw, J. L., & Iansek, R. (1995). Impaired movement sequencing in patients with Huntington's disease: A kinematic analysis. *Neuropsychologia, 33,* 365-369. doi:10.1016/0028-3932(94)00114-5

Phillips, J. G., Ogeil, R. P., & Muller, F. (2009). Alcohol consumption and handwriting: A kinematic analysis. *Human Movement Science, 28,* 619-632. doi:10.1016/j.humov.2009.01.006

Piek, J. P., Pitcher, T. M., & Hay, D. A. (1999). Motor coordination and kinaesthesis in boys with attention deficit-hyperactivity disorder. *Developmental Medicine & Child Neurology, 41,* 159-165. doi:10.1017/S0012162299000341

Pitcher, T. M., Piek, J. P., & Barrett, N. C. (2002). Timing and force control in boys with Attention Deficit Hyperactivity Disorder: Subtype differences and the effect of comorbid developmental coordination disorder. *Human Movement Science, 21,* 919-945. doi:10.1016/S0167-9457(02)00167-7

Plamondon, R. (1993). Looking at handwriting generation from a velocity control perspective. *Acta Psychologica, 82,* 89-101. doi:10.1016/0001-6918(93)90006-D

Plamondon, R. (1995a). A kinematic theory of rapid human movements. Part I: Movement representation and generation. *Biological Cybernetics, 72,* 295-307. doi:10.1007/BF00202785

Plamondon, R. (1995b). A kinematic theory of rapid human movements: Part II: Movement time and control. *Biological Cybernetics, 73*, 309-320. doi:10.1007/BF00199060

Plamondon, R. (1998). A kinematic theory of rapid human movements: Part III: Kinetic outcomes. *Biological Cybernetics, 78*, 133-145. doi:10.1007/s004220050420

Plamondon, R., Feng, C., & Woch, A. (2003). A kinematic theory of rapid human movement. Part IV: A formal mathematical proof and new insights. *Biological Cybernetics, 89*, 126-138. doi:10.1007/s00422-003-0407-9

Plamondon, R. & Maarse, F. J. (1989). An evaluation of motor models of handwriting. *Systems, Man, and Cybernetics, 19*, 1060-1072. doi:10.1109/21.44021

Plamondon, R., Yu, L., Stelmach, G. E., & Clement, B. (1991). On the automatic extraction of biomechanical information from handwriting signals. *Systems, Man, and Cybernetics, 21*, 90-101. doi:10.1109/21.101140

Poluha, P. C., Teulings, H.-L., & Brookshire, R. H. (1998). Handwriting and speech changes across the levodopa cycle in Parkinson's disease. *Acta Psychologica, 100*, 71-84. doi:10.1016/S0001-6918(98)00026-2

Ponsen, M. M., Daffertshofer, A., van den Heuvel, E., Wolters, E. Ch., Beek, P. J., & Berendse, H. W. (2006). Bimanual coordination dysfunction in early, untreated Parkinson's disease. *Parkinsonism & Related Disorders, 12*, 246-252. doi:10.1016/j.parkreldis.2006.01.006

Portier, S. J. & van Galen, G. R. (1992). Immediate vs. postponed visual feedback in practicing a handwriting task. *Human Movement Science, 11*, 563-592. doi:10.1016/0167-9457(92)90016-5

Posner, J., Maia, T. V., Fair, D., Peterson, B. S., Sonuga-Barke, E. J., & Nagel, B. J. (2011). The attenuation of dysfunctional emotional processing with stimulant medication: An fMRI study of adolescents with ADHD. *Psychiatry Research: Neuroimaging, 193*, 151-160. doi:10.1016/j.pscychresns.2011.02.005

Putzhammer, A., Perfahl, M., Pfeiff, L., Ilbach, B., Johann, M., Zitzelsberger, U., & Hajak, G. (2005). Performance of diadochokinetic movements in schizophrenic patients. *Schizophrenia Research, 79*, 271-280. doi:10.1016/j.schres.2005.05.020

Rand, M. K., Stelmach, G. E., & Bloedel, J. R. (2000). Movement accuracy constraints in Parkinson's disease patients. *Neuropsychologia, 38*, 203-212.

Romanos, M., Weise, D., Schliesser, M., Schecklmann, M., Loffler, J., Warnke, A., . . . Mehler-Wex, C. (2010). Structural abnormality of the substantia nigra in children with attention-deficit hyperactivity disorder. *Journal of Psychiatry Neuroscience, 35*, 55-58. doi:10.1503/jpn.090044

Rommelse, N. J., Altink, M. E., Fliers, E. A., Martin, N. C., Buschgens, C. M., Hartman, C. A., . . . Oosterlaan, J. (2009). Comorbid problems in ADHD: Degree of association, shared endophenotypes, and formation of distinct subtypes. Implications for a future DSM. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology, 37*, 793-804. doi:10.1007/s10802-009-9312-6

Rommelse, N. N. J., Altink, M. E., Oosterlaan, J., Beem, L., Buschgens, C. J. M., Buitelaar, J., & Sergeant, J. A. (2008). Speed, variability, and timing of motor output in ADHD: Which measures are useful for endophenotypic research? *Behavior Genetics, 38*, 121-132. doi: 10.1007/s10519-007-9186-8

Root, R., & Resnick, R. J. (2003). An update on the diagnosis and treatment of attention-deficit/hyperactivity disorder in children. *Professional Psychology: Research and Practice, 34*, 34-41. doi:10.1037/0735-7028.34.1.34

Rosenblum, S., Dvorkin, A. Y., & Weiss, P. L. (2006). Automatic segmentation as a tool for examining the handwriting process of children with dysgraphic and proficient handwriting. *Human Movement Science, 25*, 608-621. doi:10.1016/j.humov.2006.07.005

Rosenblum, S., Epsztein, L., & Josman, N. (2008). Handwriting performance of children with Attention Deficit Hyperactive Disorders: A pilot study. *Physical & Occupational Therapy in Pediatrics, 28*, 219-234.

Rosenblum, S. & Livneh-Zirinski, M. (2008). Handwriting process and product characteristics of children diagnosed with developmental coordination disorder. *Human Movement Science, 27*, 200-214. doi:10.1016/j.humov.2008.02.011

Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S. R., Simmons, A., & Bullmore, E. T. (1999). Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: A study with functional MRI. *The American Journal of Psychiatry, 156*, 891-896.

Sattler, J. M. & Ryan, J. J. (2009). *Assessment with the WAIS-IV*. La Mesa, CA: Sattler.

Schroter, A., Mergl, R., Burger, K., Hampel, H., Moller, H.-J., & Hegerl, U. (2003). Kinematic analysis of handwriting movements in patients with Alzheimer's Disease, Mild Cognitive Impairment, Depression and Healthy subjects. *Dementia and Geriatric Cognitive Disorders, 15*, 132-142. doi:10.1159/000068484

Seidman, L. J., Biederman, J., Liang, L., Valera, E. M., Monuteaux, M. C., Brown, A., . . . Makris, N. (2011). Gray matter alternations in adults with Attention-Deficit/Hyperactivity Disorder identivied by voxel based morphometry. *Biological Psychiatry, 69,* 857-866. doi:10.1016/j.biopsych.2010.09.053

Seidman, L. J., Valera, E. M., Makris, N., Monuteaux, M. C., Boriel, D. L., Kelkar, K., . . . Biederman, J. (2006). Dorsolateral prefrontal and anterior cingulated cortex volumetric abnormalities in adults with Attention-Deficit/Hyperactivity Disorder identified by magnetic resonance imaging. *Biological Psychiatry, 60,* 1071-1080. doi:10.1016/j.biopsych.2006.04.031

Semrud-Clikeman, M., Filipek, P. A., Biederman, J., & Steingard, R. (1994). Attention-deficit hyperactivity disorder: Magnetic resonance imaging morphometric analysis of the corpus callosum. *Journal Of The American Academy Of Child & Adolescent Psychiatry*, *33*, 875-881. doi:10.1097/00004583-199407000-00014

Shafritz, K. M., Marchione, K. E., Gore, J. C., Shaywitz, S. E., & Shaywitz, B. A. (2004). The effects of methylphenidate on neural systems of attention in attention deficit hyperactivity disorder. *American Journal of Psychiatry, 161,* 1990-1997.

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., . . . Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with Attention-Deficit/Hyperactivity Disorder. *Archives of General Psychiatry, 63*, 540-549. doi:10.1001/archpsyc.63.5.540

Slavin, M. J., Phillips, J. G., Bradshaw, J. L., Hall, K. A., & Presnell, I. (1999). Consistency of handwriting movements in dementia of the Alzheimer's type: A comparison with Huntington's and Parkinson's diseases. *Journal of the*

International Neuropsychological Society, 5(1), 20-25. doi:10.1017/S135561779951103X

Smits-Engelsman, B. C. M., Niemeijer, A. S., & van Galen G. P. (2001). Fine motor deficiencies in children diagnosed as DCD based on poor grapho-motor ability. *Human Movement Science, 20,* 161-182. doi:10.1016/S0167-9457(01)00033-1

Smits-Engelsman, B. C. M. & van Galen, G. P. (1997). Dysgraphia in children: Lasting psychomotor deficiency or transient developmental delay? *Journal of Experimental Child Psychology, 67*, 164-184. doi:10.1006/jecp.1997.2400

Stevens, J. P. (2009). *Applied multivariate statistics for the social sciences* (5th ed.). New York, NY: Routledge.

Stray, L. L., Stray, T., Iversen, S., Ruud, A., & Ellertsen, B. (2009). Methyophenidate improves motor functions in children diagnosed with Hyperkinetic Disorder. *Behavioral and Brain Functions, 5*, 21. doi:10.1186/1744-9081-5-21.

Swanson, J., Castellanos, F. X., Murias, M. LaHoste, G. & Kennedy, J. (1998). Cognitive neuroscience of Attention Deficit Hyperactivity Disorder and Hyperkinetic Disorder. *Current Opinion in Neurobiology, 8*, 263-271.

Teicher, M. H., Anderson, C. M., Polcari, A., Glod, C. A., Maas, L. C., & Renshaw, P. F. (2000). Functional deficits in basal ganglia of children with attention-deficit/hyperactivity disorder shown with functional magnetic resonance imaging relaxometry. *Nature Medicine*, *6*, 470-473.

Teulings, H.-L., Contreras-Vidal, J. L., Stelmach, G. E., & Adler, C. H. (1997). Parkinsonism reduces coordination of fingers, wrist, and arm in fine motor control *Experimental Neurology, 146*(1), 159-170. doi:10.1006/exnr.1997.6507

Teulings, H., Thomassen, A. J., & Van Galen, G. P. (1983). Preparation of partly precued handwriting movements: The size of movement units in handwriting. *Acta Psychologica*, 54(1-3), 165-177. doi:10.1016/0001-6918(83)90031-8

Tigges, P., Mergl, R., Frodl, T., Meisenzahl, E. M., Gallinat, J., Schroter, A., . . . Hegerl, U. (2000). Digitized analysis of abnormal hand-motor performance in schizophrenic patients. *Schizophrenia Research, 45*, 133-143. doi:10.1016/S0920-9964(99)00185-1

Tucha, O. & Lange, K. W. (2001). Effects of methylphenidate on kinematic aspects of handwriting in hyperactive boys. *Journal of Abnormal Child Psychology, 29*, 351-356.

Tucha, O. & Lange, K. (2004). Handwriting and attention in children and adults with Attention Deficit Hyperactivity Disorder. *Motor Control, 8*, 461-471.

Tucha, O. & Lange, K. (2005). The effect of conscious control on handwriting in children with Attention Deficit Hyperactivity Disorder. *Journal of Attention Disorders, 9*, 323-332.

Tucha, O., Mecklinger, L., Laufkotter, R., Klein, H. E., Walitza, S., & Lange, K. W. (2006). Methylphenidate-induced improvements of various measures of attention in adults with Attention Deficit Hyperactivity Disorder. *Journal of Neural Transmission, 113*(10), 1575-1592. doi:10.1007/s00702-005-0437-7

Tucha, O., Mecklinger, J. T., Reiter, A., Alders, G. L., Sartor, H., Naumann, M., & Lange, K. W. (2006). Kinematic analysis of dopaminergic effects on skilled handwriting movements in Parkinson's disease. *Journal of Neural Transmission, 113*, 609-623. doi:10.1007/s00702-005-0346-9

Tucha, O., Mecklinger, L., Walitza, S., & Lange, K. W. (2006). Attention and movement execution during handwriting. *Human Movement Science, 25*, 536-552. doi:10.1016/j.humov.2006.06.002

Tucha, O., Paul, G. M., & Lange, K. W. (2003). Attentional control of handwriting movements. *The International Journal of Forensic Document Examiners, 6*, 1-4.

Tucha, O., Prell, S., Mecklinger, L., Bormann-Kischkel, C., Kubber, S., Linder, M., . . . Lange, K. W. (2006). Effects of methylphenidate on multiple components of attention in children with Attention Deficit Hyperactivity Disorder. *Psychopharmacology, 185*, 315-326. doi:10.1007/s00213-006-0318-2

U. S. Food and Drug Administration. FDA Asks Attention-Deficit Hyperactivity Disorder (ADHD) Drug Manufacturers to Develop Patient Medication Guides. Retrieved from http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm107918.htm

Vaidya, C. J., Austin, G., Kirkorian, G., Ridlehuber, H. W., Desmond, J. E., Glover, G. H., & Gabrieli, G. (1998). Selective effects of methylphenidate in attention deficit hyperactivity disorder: A functional magnetic resonance study. *Proceedings of the National Academy of Sciences of the United States of America, 95*, 14494-14499. doi: 10.1073/pnas.95.24.14494

van Galen, G. P. (1990). Phonological and motoric demands in handwriting: Evidence for discrete transmission of information. *Acta Psychologica, 74*(2-3), 259-275. doi:10.1016/0001-6918(90)90008-4

van Galen, G. P. (1991). Handwriting: Issues for a psychomotor theory. *Human Movement Science, 10,* 165-191. doi:10.1016/0167-9457(91)90003-G

van Galen, G. P., Portier, S. J., Smits-Engelsman, B. C. M., & Schomaker, L. R. B. (1993). Neuromotor noise and poor handwriting in children. *Acta Psychologica, 82*(1-3), 161-178. doi:10.1016/0001-6918(93)90010-O

van Gemmert, A. W. A., Teulings, H.-L., & Stelmach, G. E. (1998). The influence of mental and motor load on handwriting movements in Parkinsonian patients. *Acta Psychologica, 100,* 161-175. doi:10.1016/S0001-6918(98)00032-8

van Roon, D., Caeyenberghs, K., Swinnen, S. P., & Smits-Engelsman, B. C. M. (2010). Children with a learning disorder show prospective control impairments during visuomanual tracking. *Research in Developmental Disabilities, 31,* 195-202. doi:10.1016/j.ridd.2009.09.004

Viviani, P. & Terzuolo, C. (1982). Trajectory determines movement dynamics. *Neuroscience, 7,* 431-437. doi:10.1016/0306-4522(82)90277-9

Wann, J. & Nimmo-Smith, I. (1991). The control of pen pressure in handwriting: A subtle point. *Human Movement Science, 10,* 223-246. doi:10.1016/0167-9457(91)90005-I

Wechsler, D. (2008). *Wechsler Adult Intelligence Scale–Fourth Edition.* San Antonio, TX: Pearson.

Whalen, C. K., Henker, B., & Finck, D. (1981). Medication effects in the classroom: Three naturalistic indicators. *Journal of Abnormal Child Psychology: An Official Publication of The International Society For Research In Child And Adolescent Psychopathology, 9,* 419-433. doi:10.1007/BF00917793

Woch, A., Plamondon, R., & O'Reilly, C. (2011). Kinematic characteristics of bidirectional delta-lognormal primitives in young and older subjects. *Human Movement Science, 30*(1), 1-17.

Yaguez, L., Canavan, A. G. M., Lange, H. W., & Homberg, V. (1999). Motor learning by imagery is differentially affected in Parkinson's and Huntington's diseases. *Behavioural Brain Research, 102*, 115-127. doi:10.1016/S0166-4328(99)00005-4

Yan, J. H., Rountree, S., Massman, P., Doody, R. S., & Li, H. (2008). Alzheimer's disease and mild cognitive impairment deteriorate fine movement control. *Journal of Psychiatric Research, 42*, 1203-1212. doi:10.1016/j.jpsychires.2008.01.006

Yeo, R. A., Hill, D. E., Campbell, R. A., Vigil, J., Petropoulos, H., Hart, B., & ... Brooks, W. M. (2003). Proton magnetic resonance spectroscopy investigation of the right frontal lobe in children with attention-deficit/hyperactivity disorder. *Journal Of The American Academy Of Child & Adolescent Psychiatry, 42*, 303-310. doi:10.1097/00004583-200303000-00010

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